Unraveling the social ecology of polio

Amarah C. Mauricio https://orcid.org/0000-0003-2131-4060
Andrew Noymer* https://orcid.org/0000-0003-2378-9860

Department of Population Health and Disease Prevention
University of California
Irvine, CA 92697-3957

18 March 2019

Abstract

Using data on poliomyelitis and typhoid fever mortality in the United States, 1914–69, we test competing theories for the twentieth century expansion of polio. We analyze data stratified by age, sex, and race. We show that some of the seemingly-paradoxical aspects of the data — principally, that whites had higher polio death rates than nonwhites but lower typhoid death rates — are consistent with the polio hygiene hypothesis. Data on racial differences show that the hygiene hypothesis is necessary and sufficient to explain patterns of polio mortality in the United States. Epidemiological phenomena are best understood in their social context.

Introduction

The *hygiene hypothesis* in polio epidemiology holds that the emergence of polio in the twentieth century stemmed, counterintuitively, from better hygiene, specifically, the increasing purity of drinking water. The explanation is that dirty water contains inoculating doses of poliovirus. As economic development made water supplies cleaner and cleaner, this inoculating function dissipated, such that exposure to poliovirus could cause frank disease (viz., paralysis). There are other "hygiene hypotheses" in medicine (f.e., Mekhaiel et al., 2011) which are not directly related.

Recent work proposed an alternate hypothesis to account for historical patterns of polio in the United States: "...contrary to the prevailing 'disease of development' hypothesis, our analyses demonstrate that polio's historical expansion was straightforwardly explained by demographic trends rather than improvements in sanitation and hygiene" (Martinez-Bakker et al., 2015,

*To whom correspondence should be addressed: noymer@uci.edu

p.1). This demographic hypothesis is a fascinating recasting of the theory of polio's expansion in the twentieth century. Coupled with results from a mathematical model, it shows that state polio incidence is correlated with birth rates, especially vis-à-vis the large postwar polio epidemics and the baby boom:

> Our results suggest the marked increase in polio incidence from the 1930s to the 1950s was a straightforward consequence of increased birth rates..., and that hygiene effects on transmission are not required to explain polio's rise to epidemic levels. (*ibid.*, p.13)

We consider the two competing polio hypotheses (hygiene, birth-rate) in light of data on white and nonwhite polio mortality in the twentieth century in the United States. We show that the birth rate explanation does not explain the observed mortality patterns. On the other hand, the hygiene hypothesis — that: "[changes in polio epidemiology] would follow a reduction in the transmission of enteric infections generated by improved sanitation and hygiene" (Nathanson and Martin, 1979, p.678) — is compatible with the mortality data.

Infectious diseases are bio-social phenomena, not exclusively biological ones (Rosenberg, 1962). Thus, it makes sense in the United States to consider race whenever the data permit it, especially given the racially-imprinted nature of American mortality (Preston et al., 2003). Diseases spread by drinking water are especially important in this regard (Troesken, 2004). This is as true today as historically (f.e., Hanna-Attisha et al., 2016). When taking racial differences into account, the hygiene hypothesis provides a parsimonious account of polio mortality in the United States. In light of racial data on mortality from poliomyelitis and typhoid fever, we show that the hygiene hypothesis has major explanatory power.

The hygiene hypothesis in polio epidemiology

The hygiene hypothesis is associated with Neal Nathanson (Nathanson and Martin 1979, Nathanson et al. 1993, Nathanson and Kew 2010). For example:

> ... the appearance of epidemic poliomyelitis was due, paradoxically, to improvements in public sanitation and in personal hygiene, both of which led to a reduction in the transmission of enteric agents such as poliovirus. A delay in the age of initial

> infection with poliovirus beyond the age when infants were protected by passively acquired maternal antibody is postulated to increase the risk of clinical disease. (Nathanson et al. 1993, p.8)

Some of the ideas that are part of the hygiene hypothesis were present in the polio literature before Nathanson's refinement, f.e., Sabin (1947), Van Riper (1947), Rivers (1948), Aycock and Meadors (1948), Paul (1952), Horstmann (1953, 1955), and Rhodes (1955). Earlier still, Collins (1946) and Melnick and Ledinko (1951) noted that in regions free of epidemic poliomyelitis, there was a steep acquisition by age of poliovirus-neutralizing antibodies, indicating high levels of exposure despite lack of outbreaks.

According to the hygiene hypothesis, improvements in sanitation set in train a series of events that led, perversely, to more, not less, disease. By "disease", we mean symptomatic (viz., paralytic) polio — not any polio infection. Poliovirus spreads via the fecal-oral route (Dömök, 1985), i.e., principally through water that is contaminated with human coliform bacteria. Poliovirus "rides along", so to say, with waterborne bacteria (Pfeiffer, 2010). First and foremost, poliovirus causes enteric disease, specifically diarrhea, although gut infection can take place even without gastrointestinal symptoms. Throughout, when we refer to subclinical cases of polio, we mean non-paralytic; many of these would have manifest as a bout of diarrhea, so were not, strictly speaking, completely free of symptoms. In a minority of cases, viremia (i.e., bloodstream infection) occurs, and in a subset of these cases, the nervous system is infected, causing some degree of paralysis, usually of the limbs (Dömök, 1985). Polio-associated paralysis may be transient or permanent. Mortality occurs when the diaphragm is affected, causing respiratory arrest.

As drinking water became cleaner, it was less likely, all else equal, that an individual would encounter poliovirus. Age is a proxy for cumulative exposure to water (and hence, to infectious doses of poliovirus). With improvements in hygiene, initial exposure to poliovirus occurred (on average) at older ages, by which maternal antibodies had waned. The cleaner the water (unless perfectly free of contamination), then the older, on average, someone would contract polio.

In the pre-vaccine era, mothers were mostly survivors of apparent or subclinical polio, themselves. Thus, their babies were born with high levels of transplacentally-acquired antibodies, known as *maternal antibodies*. In the less sanitary past, exposures to small doses of poliovirus were constant and maternal antibodies were strong. This set up a scenario such that, during the period in which they were still protected by maternal antibodies, infants and

children built up their own immune response to polio through subclinical infections. As Zinkernagel (2002) put it, "The passive transfer of maternal antibodies to the offspring at birth protects completely, then attenuates infections in the first months and nursing years by *rendering infections effectively vaccine-like*" (p.117, emphasis added); see also Zinkernagel (2001). Vaccine-induced immunity to polio can be conferred even in the presence of maternal polio antibodies (Schoub et al. 1988, Maldonado et al. 1997, Asturias et al. 2007). It stands to reason that natural subclinical infection should do the same, and that infection in the face of high levels of maternal antibodies would, indeed, be subclinical.

The equilibrium before hygienic improvements — when exposure to poliovirus was a given — was that children, as they grew older, passed through a "sweet spot." Maternal (i.e., trans-placental) antibodies protected infants from polio at birth, who were then able to develop protective immunity as these antibodies waned enough to allow subclinical (non-paralytic) infection. As the twentieth century progressed, subclinical reinfections became less common, reducing immune boosting. Across generations, this caused waning of maternal antibody at younger ages, because antibody levels were reduced in mother and therefore in child. This, in turn, narrowed the window of opportunity for infection to be "vaccine-like".

During the twentieth century, in country after country, well-documented epidemics of paralytic polio followed in the wake of improvements in drinking water. Payne (1955) writes: "Within the last decade or so, many countries have experienced relatively serious outbreaks of poliomyelitis for the first time"; see also Sabin (1949) and Candioti et al. (1949). This is consistent with the hygiene hypothesis and applies to a diverse set of countries at different stages of their demographic transitions (on which, cf.. Kirk, 1996). Infection of travelers to countries in which there was no epidemic polio (May 1950) indicates that poliovirus had a worldwide distribution despite not showing an epidemic pattern in all regions; a similar effect can be seen with expatriate Europeans living in Casablanca, compared to Moroccans (Paul and Horstmann, 1955). Countries in the pre-epidemic phase had not yet crossed the threshold of sufficient sanitation to permit polio epidemics (Paul 1958, Bunimovich-Mendrazitsky and Stone 2005). At least in the United States, water quality improvement was a gradual, place-by-place process (Meeker, 1971; Condran and Crimmins-Gardner, 1978; Cutler and Miller, 2005).

Figure 1 shows a classic finding by Paul and Horstmann (1955), of antibody prevalence in Casablanca, Morocco, in 1953. At birth, effectively all

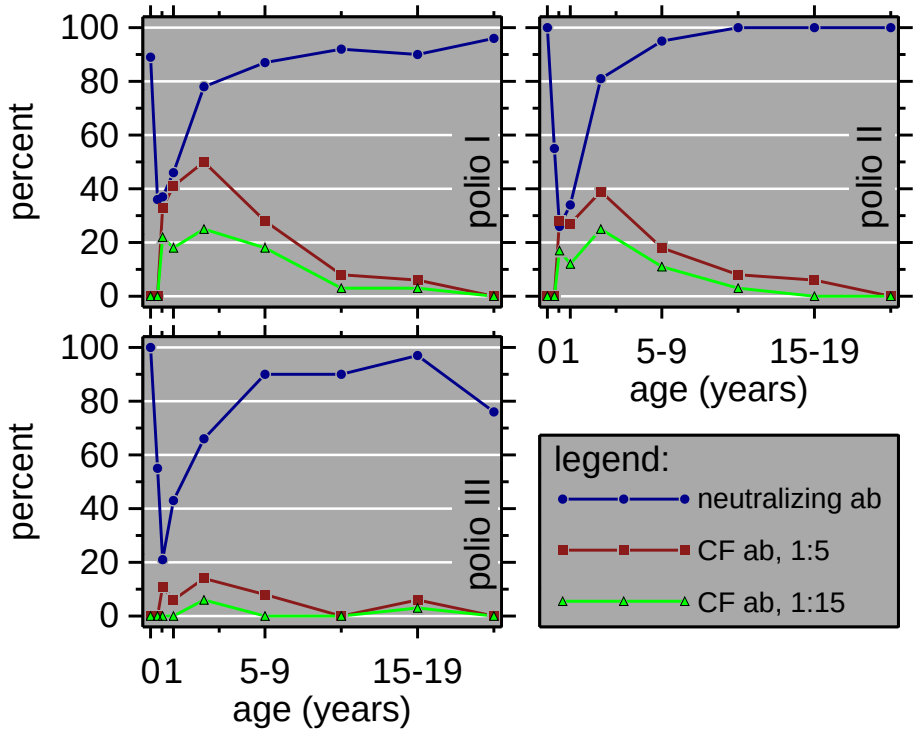

Figure 1: Antibody ("ab") prevalence by age, Casablanca, Morocco, 1953. Clockwise from upper left: polio type I, polio type II, legend, and polio type III, as labeled; "CF": complement fixing at stated dilution. Data from Paul and Horstmann (1955).

infants have neutralizing (maternal) polio antibodies, which decline during infancy. Neutralizing antibodies return during the childhood years, as a result of (subclinical) poliovirus infection "without a high incidence of the recognized paralytic disease" (*ibid.*, p.522). The compliment fixing antibody curves show the age pattern of relatively-recently acquired polio immunity (approximately, the prior 36 months), and are consistent with the pattern seen in the persistent neutralizing antibodies. The protection afforded by antibodies is mostly homotypic (Nathanson and Martin, 1979; Dömök, 1985), although early assays did not have excellent specificity among the three types (Melnick et al., 1954).[1] These are cross-sectional data, so their in-

[1]To the extent to which protection is heterotypic, type 2 polio infection may protect against type 1 infection, but not vice versa or with respect to type 3 (Hammon and Ludwig, 1957).

terpretation in terms of cohort experiences requires assuming a stable equilibrium, which is reasonable given the lack of outbreaks of paralytic disease. Similar findings for Tahiti are shown by Horstmann et al. (1955) and Kessel et al. (1956), for Cairo, Egypt by Goldblum and Melnick (1952) and Paul et al. (1952*a*), for Alaska natives by Paul et al. (1952*b*), and for some South American and Caribbean populations by Melnick (1959). Turner et al. (1950) also compile similar data for several populations (see especially their figures 6 and 7).

With improvements in sanitation, the first dose of poliovirus was more likely to be in a completely immuno-naïve individual, so was more likely to produce severe disease. Changes in birth cohort sizes are absent from the hygiene hypothesis, because they are not necessary for it. As societies became more hygienic, the force of infection of polio lowered, shifting the age distribution of infection upwards (on the force of infection and its inverse relation with the mean age of infection, cf. Grenfell and Anderson, 1985). The age effect was so profound, that contemporary observers attributed enormous importance to age-severity relationships (Horstmann, 1955, 1963; Nathanson and Martin, 1979). However, age is not the key factor in the hygiene hypothesis. Except as regards crossing the threshold of maternal antibody protection, age matters only because it is a proxy for cumulative exposure to polio virus. Moreover, following hygienic improvements, the average age of infection increases over time partly through cohort effects. For instance, suppose, hypothetically, that (mostly) clean water is imposed all-at-once. Ten years later, most ten year-olds will be susceptible to polio, whereas before, most were immune. After five more years, most fifteen year-olds will be susceptible to polio (modulo those who were infected and recovered, or died, in the interval), and so on.

Hygiene hypotheses

The polio hygiene hypothesis suggests the following sub-hypotheses, relative to the United States' experience:

1. As water supplies became cleaner, polio death rates at ages above infancy should *increase*, until the advent of the inactivated poliovirus vaccine (IPV) in 1955 (Francis and Korns, 1955; Francis, 1955).

2. Polio mortality rate increases seen during the twentieth century should be more profound in people above age 5 than in infants and children.

3. Nonwhites, as a group, had inferior access to clean water. Therefore, polio death rates among nonwhites should:
   (a) increase less over the twentieth century, compared to whites;
   (b) be lower than among whites, except during infancy;
   (c) decline in the post-vaccine era (assuming equal access to IPV).
4. Apart from general considerations of sex differences in mortality (f.e., Ciocco, 1940*a*,*b*; Nathanson, 1984), these patterns (in 1–3) should be the same by sex.

These derive from the hygiene hypothesis and rest on the principle of mortality rates as an indicator of severe disease. Regarding 3b, note that before vaccine-protected mothers started having children themselves, most infants were born with high levels of maternal antibodies (Tanzi et al., 1997). Therefore, to die of polio in infancy meant exposure to a large enough inoculum to overcome maternal antibodies, which we expect to occur more often among those drinking less clean water (viz., nonwhites, as we will demonstrate from typhoid fever mortality).

Data

To test these hypotheses, we examine polio death rates by age, race, and sex in the United States Death Registration area, 1914–32, and in the United States, 1933–69. For simplicity, we refer to the data for the entire time span, 1914–69, as being for the United States. Prior to 1933, the data we use do not cover the entire country (Hetzel, 1997, pp.43–66). Because of our racial-comparative approach, the incompleteness of death registration prior to 1933 is not a major obstacle; the area being compared is always the same (viz., across races, holding year constant). The data sources are listed in Appendix A (p.25). To the best of our knowledge, there are no published data on age-, race- and sex-specific polio death rates prior to 1914. The impact of vaccination necessitates stopping our analysis in 1969. In the late 1960s, polio death rates were less than one per million in our demographic groups, and some years had zero deaths in some age/sex/race cells. The death rates we examine are racially categorized as white/nonwhite, with no finer granularity available in the source data. In this time period, the nonwhite population of the United States was overwhelmingly black or African-American (Lerner, 1975).

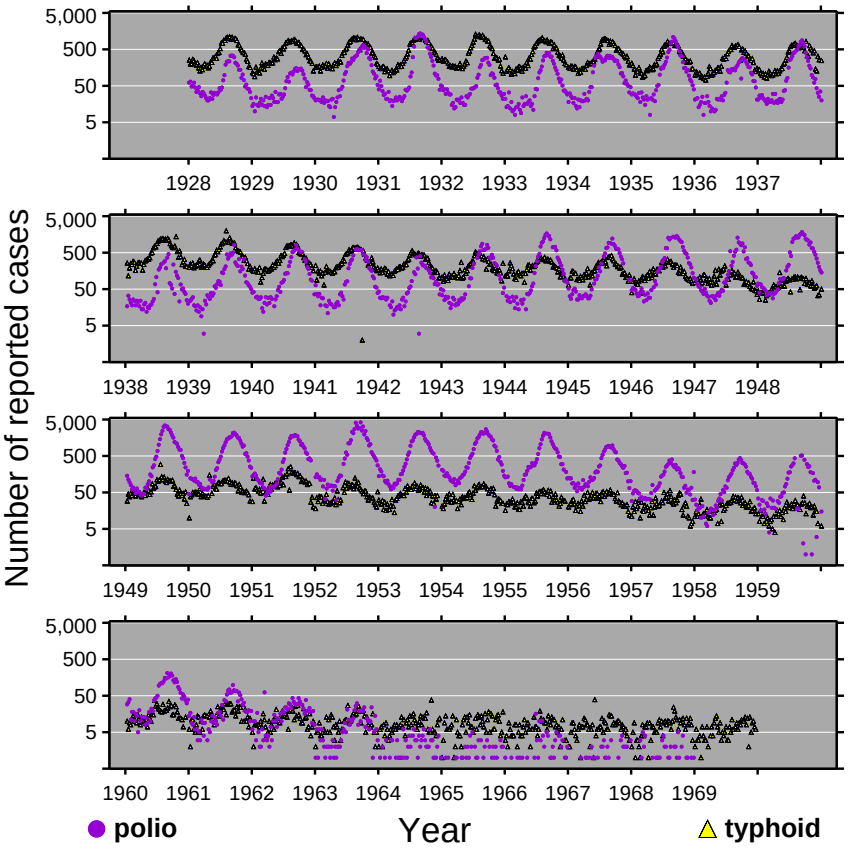

Figure 2: Weekly polio and typhoid fever incidence, United States, 1928–69. Data source: Project Tycho (2016).

The key aspect of our analysis by race is that nonwhites were drinking dirtier water. We use typhoid fever as a comparison disease because "the typhoid-fever death-rate of the community will fairly well represent the sanitary quality of the water-supply" (Whipple, 1907, p.7); cf. also Whipple (1908), p.77. For more recent use of typhoid death rates as an index of water quality, see Higgs and Booth (1979), Ewbank (1987), Troesken (2001), Condran and Lentzner (2004), Cutler and Miller (2005), Ferrie and Troesken (2008), and Beach et al. (2016). Typhoid vaccination had a negligible impact on mortality (Collins and Councell, 1943). Both poliovirus and the typhoid fever pathogen (the bacterium *Salmonella enterica* seroytype typhi, sometimes called *S. typhi*) are spread by the fecal-oral route (i.e., principally

through contaminated drinking water), which makes typhoid an appropriate comparison disease. Figure 2 is a time series plot of weekly incidence (not mortality) of typhoid fever and poliomyelitis in the United States, 1928–69, using data from Project Tycho (2016). The absolute levels are difficult to interpret, for two reasons. First, the data are subject to reporting-area changes over time (van Panhuis et al., 2013), and as a result, these are reported cases, not rates. Second, especially for polio, these are systematically underreported. For every case of clinical (i.e., paralytic) polio there are 100–1,000 inapparent infections (Melnick and Ledinko 1953, Horstmann 1963). Subclinical infection occurred in up to 70% of household contacts of polio cases (Isacson et al., 1957). Figure 3 demonstrates that both of these waterborne diseases have the same seasonal pattern, so are highly appropriate comparison diseases. The seasonality in figure 3 coheres with contemporary detailed virological surveillance (Honig et al., 1956). Where typhoid and polio differ is that reinfection can occur with typhoid fever (Parry et al., 2002), whereas polio infection confers lifelong immunity. For this reason, typhoid fever death rates do not decline as steeply with age.

Results

Typhoid fever

Figure 3 shows typhoid fever mortality in the United States, 1914–69. It contains time series plots of typhoid death rates, separately by sex and by age groups: 0, 1–4, 5–14, 25–34, and 35–44 years. Within each age/sex panel, the races (white, nonwhite) are plotted independently. Throughout this period, at age 45 and above, polio death rates (to which these typhoid data will be be compared) were extremely low among all race/sex combinations — on the order of 1 death per million population, or less. Thus, the age groups from which meaningful comparisons may be drawn are shown in figure 3 and in figure 5 (for polio). Appendix B (p.26) gives some descriptive statistics of these data (tables 1–6), and explains the gaps in the series. Tables 7–12 (also in appendix B) summarize tests of statistical significance of white/nonwhite differences. Throughout, we use $\alpha = 0.05$ as the significance level (type I error rate).

The data in figure 3 are quite straightforward. Typhoid fever death rates show a consistent decline throughout the twentieth century before virtually vanishing in the 1950s and ’60s due to a combination of clean water and antibiotic drugs. Nonwhite typhoid death rates are consistently higher than

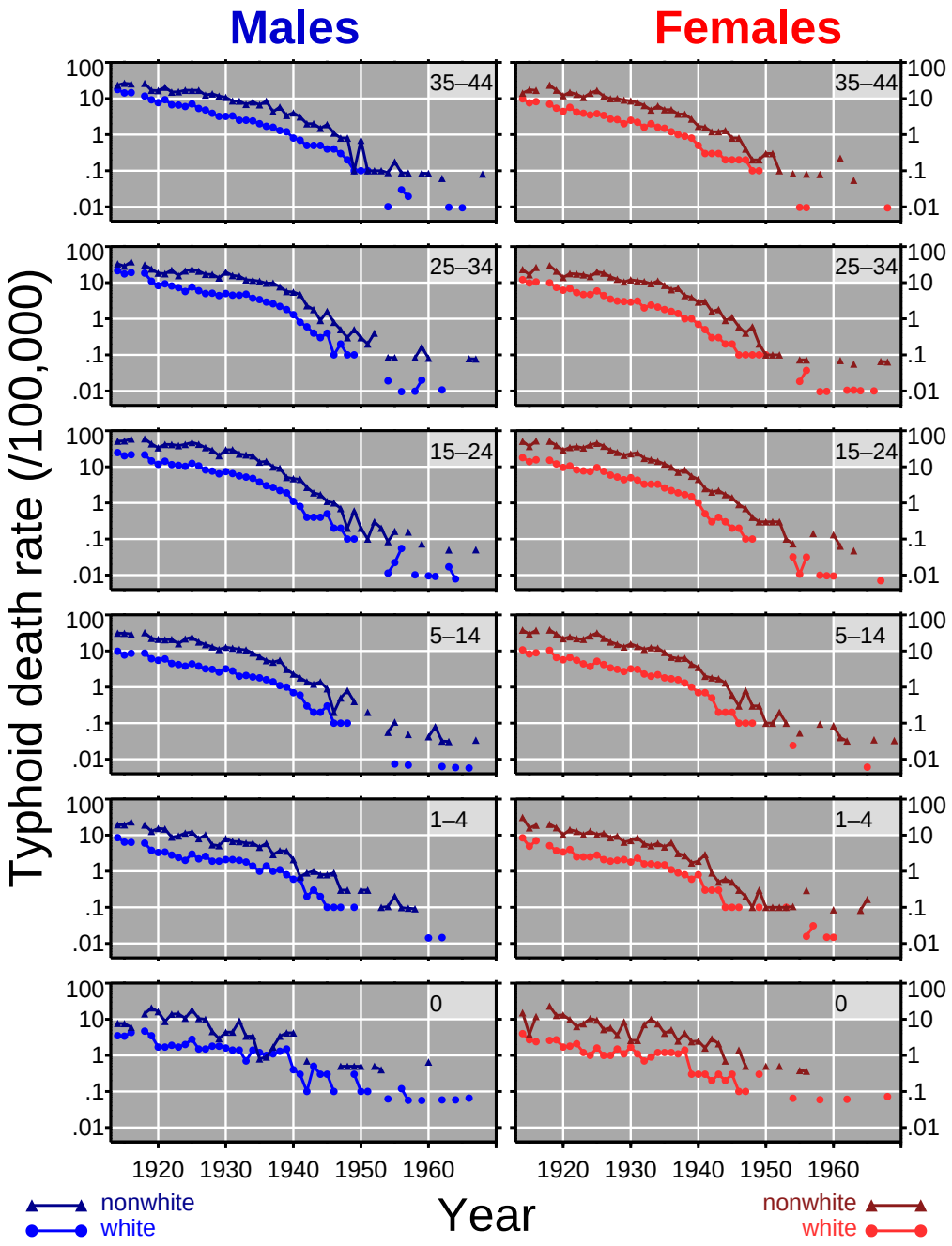

Figure 3: Typhoid fever death rates, United States, 1914–69, ages 0–44, disaggregated by 6 age groups, sex, and race

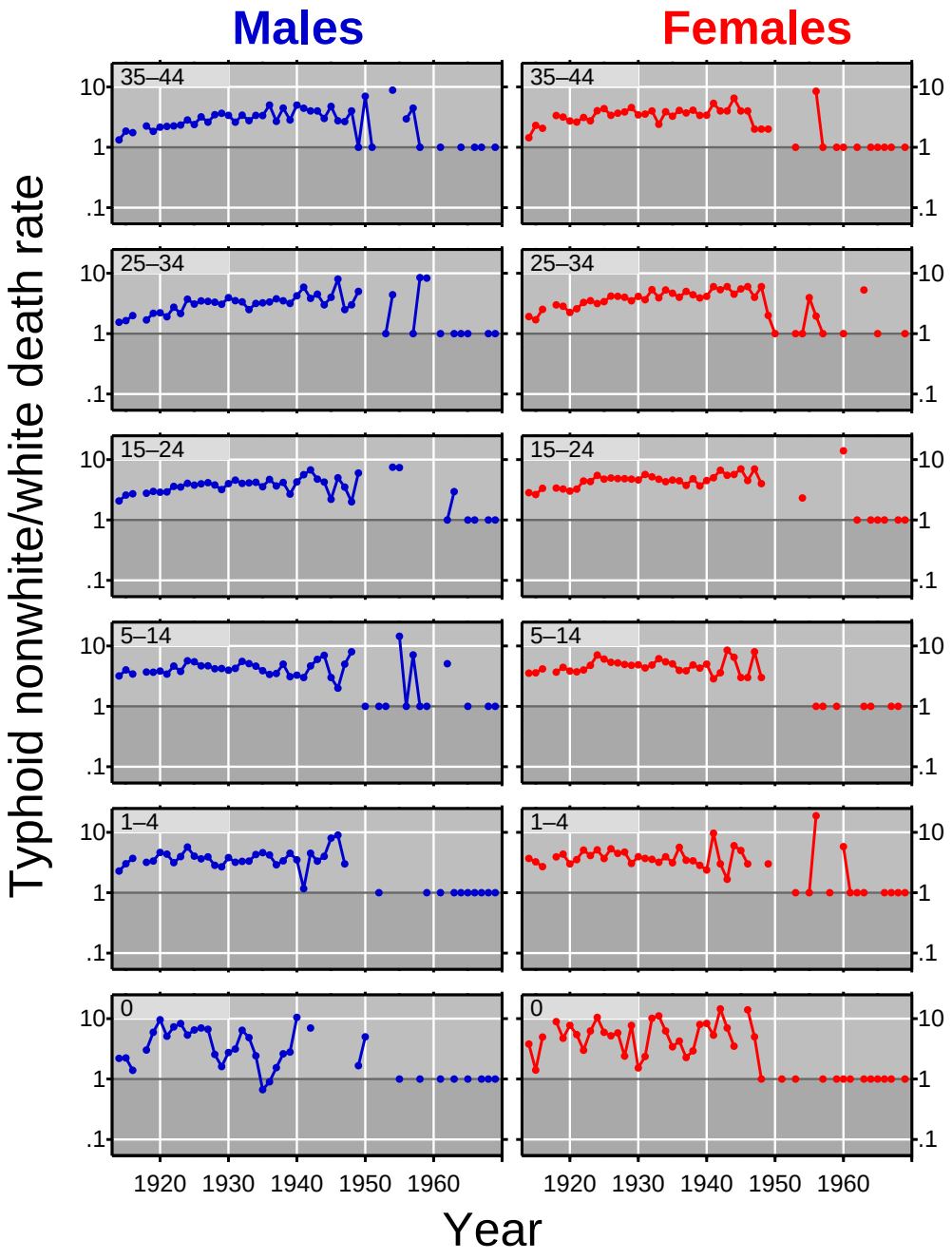

Figure 4: Ratio of nonwhite to white death rates, typhoid fever, United States, 1914–69, ages 0–44, disaggregated by 6 age groups, sex, and race. A number of missing values are generated due to division by zero. Cells in which both whites and nonwhites are zero have been forced to 1.0 (see f.e. 1960s).

those for whites. These racial gaps are statistically significant in most years (Appendix B, tables 7–12). There are some years in which the differences are non-significant, especially in years in which there are few deaths. There are no years in which whites have higher typhoid death rates in a statistically-significant way, but many years in which nonwhites' excess is statistically significant.

Figure 4 shows the same data, recast as the nonwhite/white death rate ratio. This visualizes striking racial differences: sometimes a 10× higher typhoid fever death rate for nonwhites. These results are consistent enough that the distinction between ratio or difference is not important (cf. Sheps, 1958, 1959). Figures 3 and 4 amply demonstrate that nonwhite Americans had inferior access to clean drinking water in this time period (Troesken, 2001). Blacks living in the rural south were drinking dirtier water, in many cases because they lacked piped water, tout court (Cowhig and Beale, 1964*a*,*b*). No typhoid coming in through pipes is no guarantee that alternate water sources are clean, and the difficulty of hand-washing in such situations enhances person-to-person typhoid transmission (as well as that of poliovirus, Dömök 1985).

Poliomyelitis

Having shown that nonwhites were drinking dirtier water, we turn here to the main analysis: poliomyelitis mortality. Figure 5 shows polio death rates in the United States, 1914–69. A number of patterns are noteworthy. Particularly at younger ages, the 1916 polio epidemic in the northeast, which struck New York especially hard (Lavinder et al., 1918; Trevelyan et al., 2005*b*), can be seen clearly. The national data mask the epidemic nature of polio. For example, if there is an epidemic one year in Chicago, the next year in Milwaukee, in San Antonio the year after that, and so on, the aggregate time series picture does not necessarily look like one of outbreak/quiescent cycles (Gilliam et al., 1949*a*,*b*; Trevelyan et al., 2005*a*).[2]

Of course, the entire pattern of poliomyelitis mortality can be regarded as a fifty-year epidemic. Consider, especially, the series in figure 5 for whites, ages 25–34 — which rose slowly and then came crashing down upon the intervention of the inactivated poliovirus vaccine (IPV) in 1955. Indeed, "it is rare for a serious disease to be controlled so quickly and dramatically as was poliomyelitis" (Melnick, 1993, p.191). The increase in polio mortality

[2]This is well studied with measles, f.e., Bartlett (1957); Murray and Cliff (1977); Leeuwenburg et al. (1979); Cliff et al. (1992*a*,*b*).

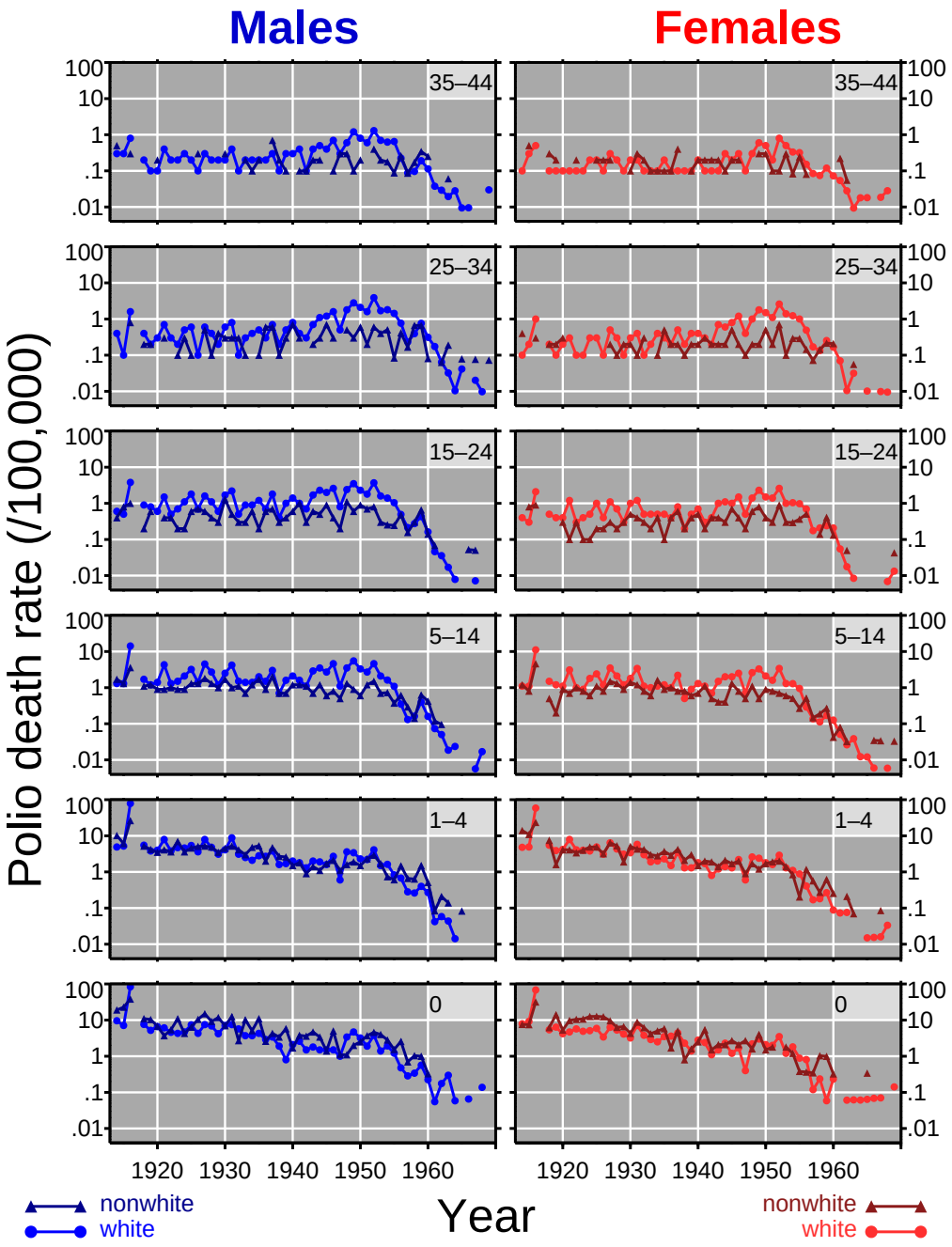

Figure 5: Polio death rates, United States, 1914–69, ages 0–44, disaggregated by 6 age groups, sex, and race

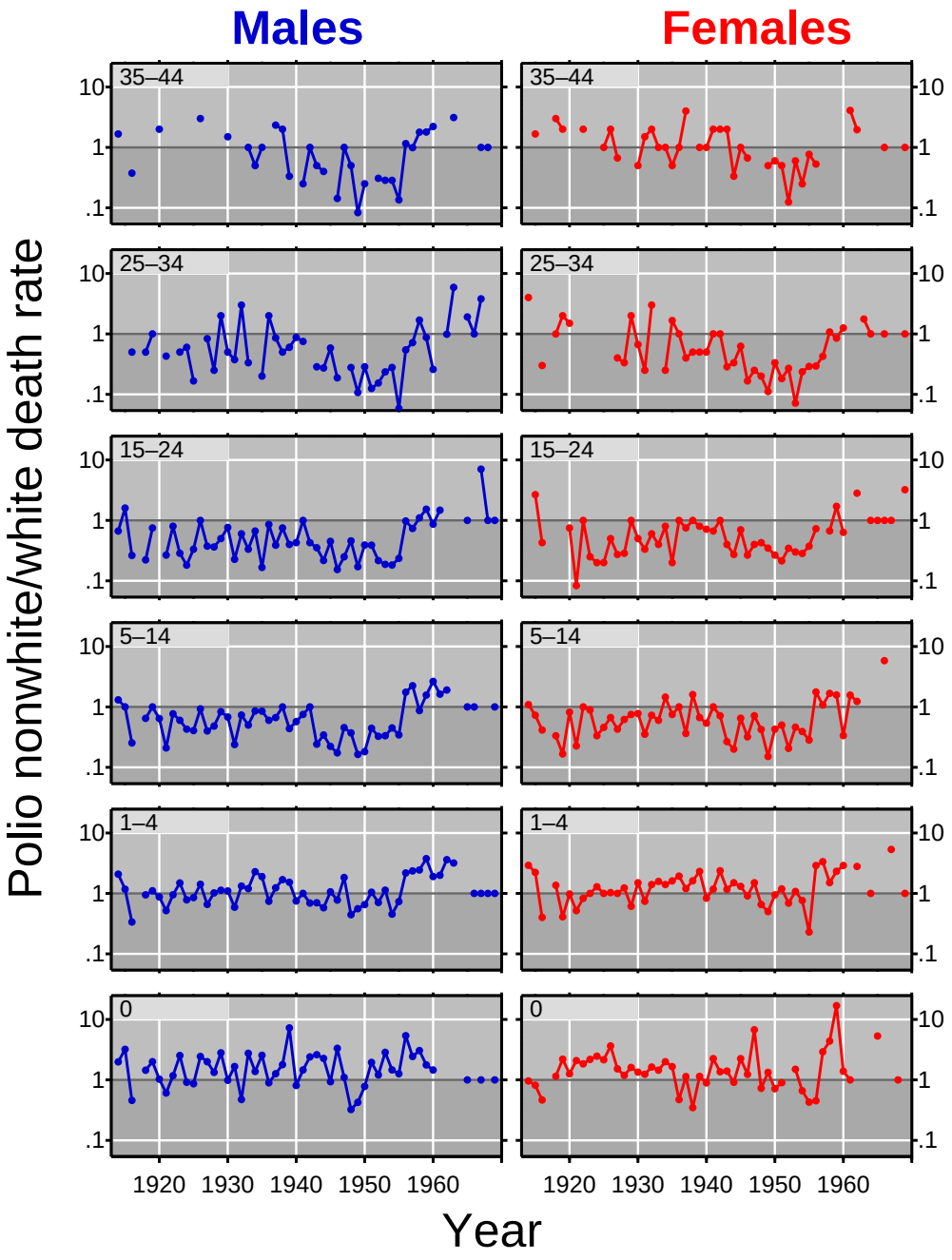

Figure 6: Ratio of nonwhite to white death rates, polio, United States, 1914–69, ages 0–44, disaggregated by 6 age groups, sex, and race

among whites in the 1940s and early '50s, was socially significant (Rogers, 1992, 2014; Oshinsky, 2005); it was, after all, the post-penicillin era, in which the expectation was that infectious diseases should be vanquished. Consider also the remarkable pattern that white polio death rates in the early 1950s didn't decline with age until the 35–44 age group (discussed further, below).

The racial comparisons for polio are very different than those for typhoid. Figure 6 shows that despite (or, indeed, because of) drinking cleaner water, whites had higher polio death rates than nonwhites for most observations above infancy. Differences post-1955 have to do with IPV. For instance, death rates are lower for nonwhites in the 5–14 age group for virtually the whole period, 1914–1955 (figure 6) and then reverse after the mid 1950s. We attribute this to differential access to vaccination. We have little data on polio vaccination rates by race, but what documentation we have found shows that lower socioeconomic groups had lower vaccination rates (Melnick et al., 1961, p.1160).[3] Appendix C (p.40) shows that the nonwhite/white mortality ratio seen for typhoid fever is (broadly) similar to that seen for other infectious diseases (except polio), underscoring how unusual the polio patterns are from a racial perspective.

Age-mortality profiles

The data in figures 3 and 5 can be re-visualized as age-mortality profiles (plots with age on the horizontal axis and mortality rates on the vertical axis). This is done in figures 7 (typhoid) and 8 (polio); the vertical axis is inverse hyperbolic sine-transformed.[4] Each panel of figures 7 and 8 shows one race$\times$sex combination. To illustrate the evolution, four years (1920, '30, '40, '50) are shown. Another way to compare age-mortality profiles is to use a heatmap; these are presented using all years in Appendix D (p.42).

The patterns for typhoid (figure 7) are straightforward. Each mortality profile is nested beneath the chronologically-previous one, with the slight exception of 1920/30 for nonwhite males. These graphs show progress, in which the age-mortality profiles sink over time. For whites, by 1950, typhoid fever death rates were zero or nearly zero for all ages. For nonwhites, there is also decline, but the age-mortality profile in 1950 is not flat, although it has changed shape relative to the earlier years. The 1950 curve for nonwhites is convex, having maxima at the extremes of age, while those for 1920 and

[3] Better-documented is the unfavorable access to measles vaccination for nonwhites, in the period after its introduction in 1963 (Dandoy, 1967; Pyle, 1973).

[4] This is a log-like transformation that preserves zeros (Burbidge et al., 1988).

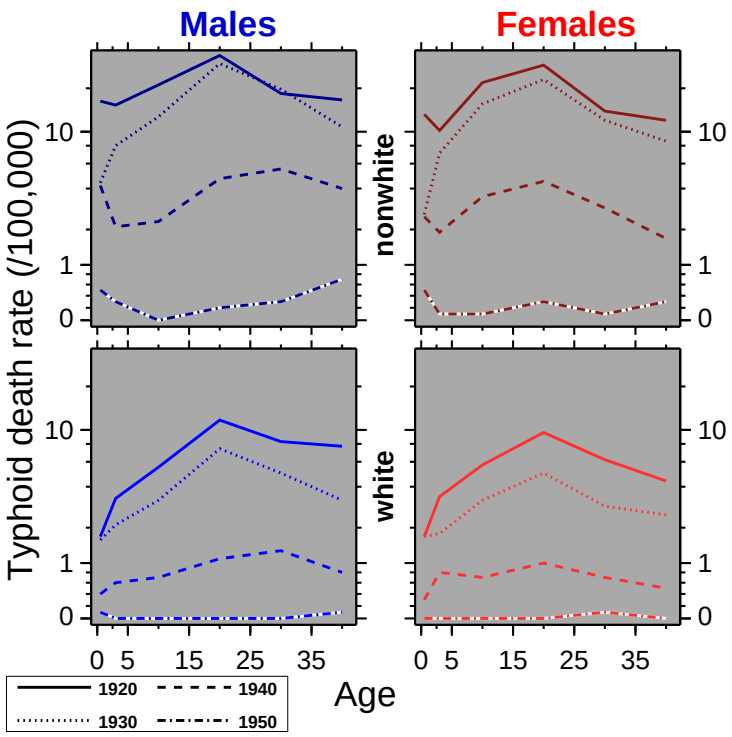

Figure 7: Age-mortality profile, typhoid fever, United States; 1920, '30, '40, '50; ages 0–44 (see legend); by sex (columns), and race (rows).

1930 are concave, having a maximum in the 15–24 age group; 1940 has both convex and concave sections. The nonwhite age mortality profiles reflect reduced access to clean water compared to whites, persisting even to 1950. What is more — since these are death rates, not incidence — the nonwhite mortality may reflect less access to antibiotics, especially chloramphenicol after 1948 (Hornick, 1992). Earlier antibiotics proved less effective against typhoid fever (Parsons, 1948). Although work on typhoid fever vaccines goes back to the nineteenth century (Gröschel and Hornick, 1981), these are not regarded as having played a role in the decline of typhoid mortality at the population level (McCaw, 1919; Levine and Blake, 1992).

The relatively flat age-mortality profiles for typhoid (i.e., compared to polio, figure 8) reflect the lack of long-lasting immunity. Children show lower typhoid mortality than adults in figure 7. This may be due to misattribution of typhoid deaths among children (Troesken, 2004, p.28). Alsan and Goldin

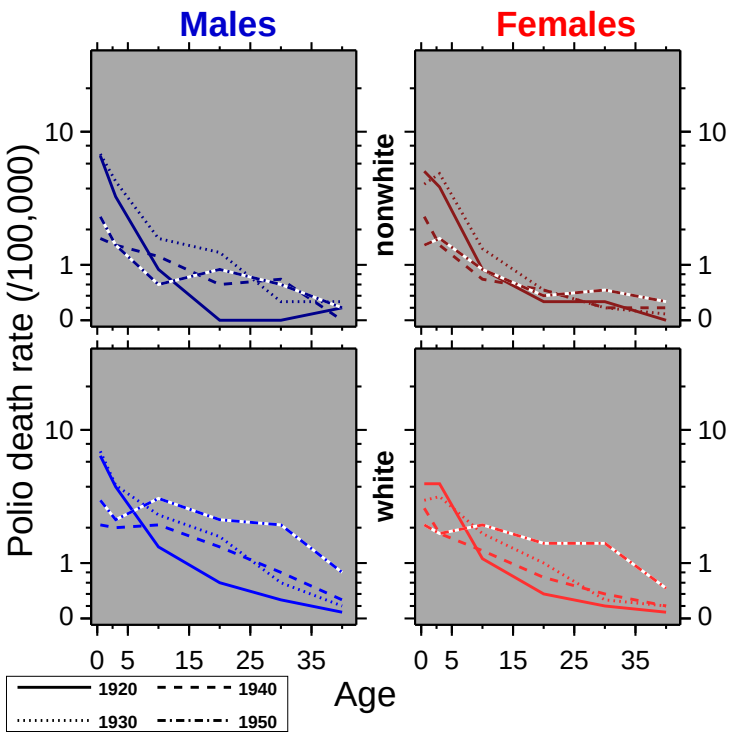

Figure 8: Age-mortality profile, poliomyelitis, United States; 1920, '30, '40, '50; ages 0–44 (see legend); by sex (columns), and race (rows).

(2018) show declines in all-cause child mortality when water was improved, which is consistent with the misattribution hypothesis. Alternatively, milk pasteurization could have had more positive effects on children than adults. The extent to which pasteurization lowered typhoid death rates is unclear (Frost, 1916; Sydenstricker, 1928). Adults had higher typhoid death rates than children going back to 1900 if not before (U.S. Department of Health, Education, and Welfare, 1956). This, again, argues for the classification problems, not a massive impact of pasteurization. In any case, the take home message from figure 7 is that, regarding typhoid, things got better over time.

Reflecting the complex epidemiology of poliomyelitis, the patterns in figure 8 are more complicated. The discrimination between the curves is harder because they are overlapping, not nested. First, consider the age-mortality profiles for polio for 1920 (solid line pattern in figure 8). The earliest data series in this figure is also the lowest — this is not the picture of progress

as seen with typhoid. With polio, the earliest (1920) age-mortality profiles are the easiest to explain. Death rates declined steeply by age in 1920, in all demographic groups. Water was (comparatively) dirty, so even infants with maternal antibodies could become infected. At older ages, death rates declined because these individuals grew up with even dirtier water, and most had acquired immunity to polio (not all, as death rates were still above zero, except nonwhite males in 15–24 and 25–34 year age groups). Survival to older ages selected for individuals with upbringings in higher socioeconomic status households. This selection also is for greater susceptibility to polio, which is partly why polio death rates are not even lower at older ages in 1920. See Kurland et al. (2009) and Zajacova and Burgard (2013) on this type of mortality selection.

For the later polio age-mortality profiles, consider them separately by race. For whites, there are declines over time of polio death rates for infants and children. Cleaner water is less capable of "punching through" infants' maternal antibodies. As water improved, mortality rates in this age group fell over time. The age-mortality profiles after 1920 do not decline by age anywhere near as steeply as in 1920. The 1930 and 1940 curves are quite similar, indicating stalled progress against polio.

The most profound feature of figure 8 is how the 1950 curve is the highest (i.e., worst) for most ages — for whites only (bottom two panels). Polio in the pre-vaccine era was sometimes called "infantile paralysis" (Paul, 1971). Yet, mortality rates at the population level among whites were as high or higher for 5–14 year-olds as they were for infants.[5] Indeed, by 1950, the polio death rates among whites are virtually a plateau by age, with the most profound declines not occurring until the 35–44 age group.

The top two panels of figure 8 are for nonwhites. Females (upper right) show the pattern which is easiest to interpret. Polio mortality for nonwhite infants and girls fell over time, particularly considering 1920 and 1930 (together) as one time step, and 1940 and 1950 as another. This is because by 1940 water improvements (on average) had belatedly come to the nonwhite population, so poliovirus was less able to puncture partial immunity, as discussed above. For older nonwhites (i.e., above age 5 for 1920 and 1930, or above age 15 for 1940 and 1950), polio death rates are low because members of these cohorts grew up with dirty water, and were immune. The pattern for nonwhite males is a little more complex, with 1920 having unambiguously the lowest death rate at older ages. The age-mortality profiles for nonwhite

[5]For males: 3.3 deaths per 100,000 population for 5–14 year olds vs. 3.2 for infants; for females, 2.1 for both age groups.

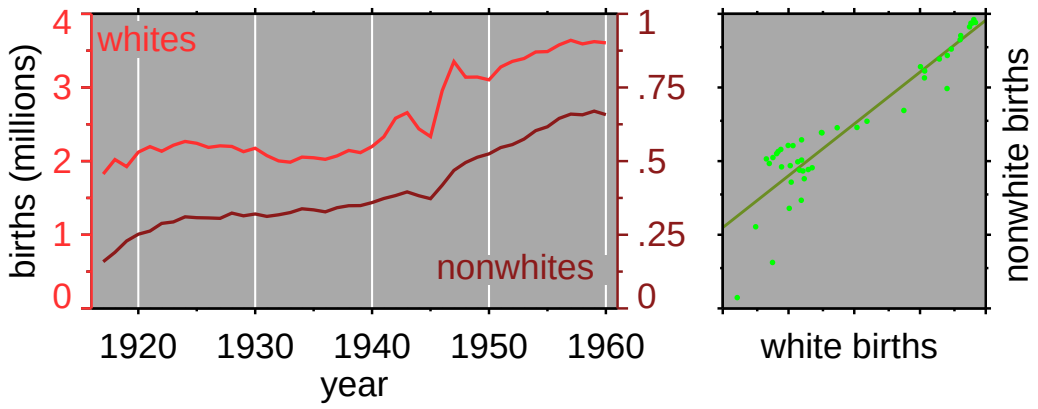

Figure 9: Left panel: births, white (left y-axis) and nonwhite (right y-axis), United States, 1917–60. Right panel, elasticity plot (log-log scatterplot, with regression line), white and nonwhite births.

males resembles those of white males a little more than nonwhite females vis-à-vis whites. Nonetheless, nonwhites show the expected pattern of a population drinking dirty water: polio death rates are lower in adults than in infants and children.

The postwar baby boom

If the baby boom catalyzed the increase in polio in the 1950s, we should expect divergent patterns in the increase in births, by race. Figure 9 (left panel) gives annual births in the United States, 1917–60, by race. The baby boom is clearly visualized, and occurs for both racial groups. The right panel of figure 9 is a log-log plot of nonwhite versus white births; the slope of the regression line is the elasticity (Greene, 1997, p.227), or the percent change in nonwhite births for a percent change in white births. The data are a good fit to a straight line ($R^2 = 85.5\%$), affirming that the baby boom was not a whites-only phenomenon.

Figure 10 presents another scale-free way to depict the changes in white and nonwhite births. This is a time series of the year-on-year percent change in births, by race.[6] The two racial groups are in good synchrony according

[6]This is $\log(B_{Y+1}/B_Y)$, in which B is the number of births and the subscript Y denotes year.

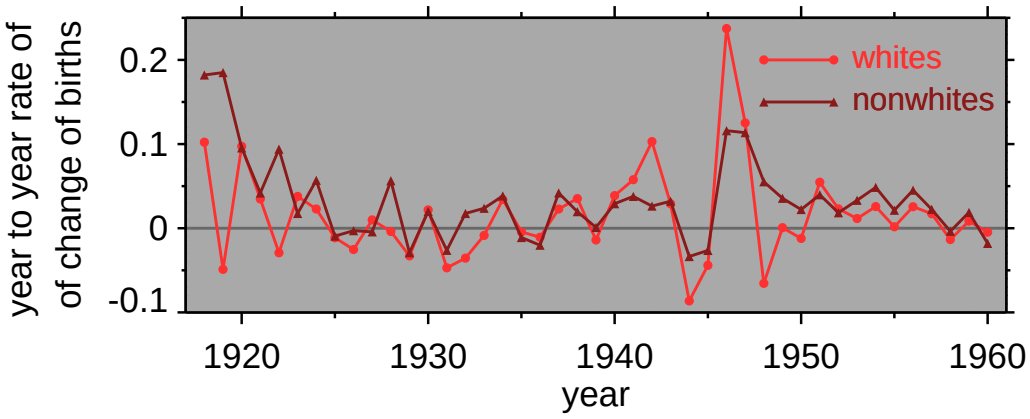

Figure 10: Year-on-year rate of change in number of births, United States, 1918–60.

to this measure. The white baby boom was a bit more of a percussion, with 1946 having over 20% more births than 1945, compared to just over 10% for nonwhites. On the other hand, in 1948 compared to '47, whites showed a decline in births whereas the baby boom for nonwhites continued. The picture from figures 9 and 10 is that — modulo some small differences — both whites and nonwhites experienced a baby boom of similar timing and similar proportional size.

Discussion

Nonwhites consistently had dirtier water, as shown by figures 3 and 4 which give absolute and relative typhoid mortality rates by race. Typhoid fever is a waterborne infection for which the hygiene hypothesis does not apply because there is no lasting immunity. Consider especially figure 4, where the data are always (except two points) on or above the line of equality (i.e., nonwhite mortality rates greater than or equal to those for whites). Moreover, many of the values at 1.0 in figure 4 denote equal values because death rates are zero for both racial groups (i.e., 0/0 is treated as equality). For typhoid, there are two years (per sex) in which the nonwhite/white ratio is 1.0 but the underlying values are not zero.

Compared to typhoid, the polio mortality data in figures 5 and 6 show a different pattern over time and across ages and races. Focus on figure 6, the

white/nonwhite ratio of poliomyelitis mortality rates. Among infants (age "0" in figure 6), nonwhites tend to have excess mortality. Infancy is the age at which maternal antibody protection predominates (cf. figure 1). In infancy, we don't expect the hygiene hypothesis to hold, because not enough time since birth has elapsed to accumulate inoculating exposures. Infection at this age can be "vaccine-like" (Zinkernagel, 2002, p.117), setting up permanent lifetime immunity (figure 1), all the more so in the presence of later exposures to poliovirus, which act as boosters. If water is dirty enough, it can overwhelm passive maternal-antibody protection, causing frank poliomyelitis, and potentially death. In infancy — and infancy only — we expect those drinking dirtier water to have higher polio mortality rates; this is what the data reveal. For polio, 16 of the 28 instances of a ratio of 1.0 (for males) and 13 of 35 (for females) in figure 6, represent zero divided by zero.

Next, consider polio mortality at ages 5 and over (the top four age-group panels of figure 6). Here we see clear evidence of white excess polio mortality (ratio below 1.0). Before 1940, the data are somewhat sparse for 35–44 year-olds. These cohorts were born in the late nineteenth and early twentieth centuries, yielding small sample sizes of polio deaths at advanced ages; most everyone acquired immunity in childhood. Another salient pattern is that the white excess becomes more profound over time, especially after 1940, but only up to the mid 1950s. This is a reflection of white cohorts growing up with cleaner water (Troesken, 2004; Cutler and Miller, 2005), and therefore being susceptible to polio at ages 5 and above. The sudden shift to nonwhite excess toward the end of the data series is due to unequal access to the vaccine. The double effect of further water quality improvements and of vaccination results in the sparse data in the 1960s. Since maternal antibodies do not vanish on the first birthday, the data for ages 1–4 show a pattern intermediate between infants and the 5–14 age group.

The typhoid data show that nonwhites were not drinking cleaner water. Is it possible that clean water as regards typhoid and polio are two independent phenomena? Suppose, for the sake of argument, there was less polio transmission among nonwhites, and that this was maintained by, say, residential segregation. In this scenario, nonwhites could have lower mortality rates for polio, while typhoid epidemiology would exist in its own compartment. Melnick et al. (1955, 1957, 1962), and Le Bouvier (1957) show that lower socioeconomic status (SES) residents of Phoenix, Arizona and Charleston, W. Virginia had higher levels of polio antibodies than their higher SES counterparts. In addition to the mortality data for infants, this is strong evidence against the hypothetical that there was less polio transmis-

sion in nonwhite communities. Melnick and Parks (1964) present similar data for Houston, Texas, but during the early vaccine era, so the data are not completely comparable.

Nothing in the flow of white and nonwhite births (figures 9 and 10) would seem to predict the racial differences in polio mortality. The dynamics of infectious diseases — especially those in which there is post-recovery immunity — are intimately related to birth rates, which provide a flow of susceptible individuals. Epidemics reduce the stock of susceptibles, which must then be replenished by new flows, and so on (Anderson and May, 1991). Given that the baby boom began in 1946 (Easterlin 1961, Healy 2018), there is no doubt that it somehow affected polio epidemiology during the "major epidemic years (1949–1954)" (Eiben, 1997, p.108). Given the general similarity of the white and nonwhite baby booms, it is hard to see how the births could have governed the divergence of mortality patterns by race. What is more, "thirty-five percent of [polio] patients admitted... [in 1949–1954] were adults" (*ibid.*), which is much more consistent with the hygiene hypothesis.

Viewing the mortality patterns through a cohort lens helps bring them in into focus (Ryder, 1965; Hobcraft et al., 1982). Fifteen to twenty-four year-olds were born 15–24 years ago, etc. Their polio mortality patterns reflect current circulation of poliovirus in the water supply, but also their immunity as a cohort. The latter is influenced by poliovius circulation when they were infants, 15–24 years ago, as well as in the intervening years. Similarly, *mutatis mutandis*, for other birth cohorts. Before the vaccine, drinking tap water was a risk factor for polio, except in cases where water filtration and chlorination worked absolutely perfectly. Cohorts which grew up before substantial improvements in water quality were overwhelmingly immune to poliomyelitis as adults. Significant improvements in tap water quality by the mid-1920s (for whites) — not the baby boom — set the stage for the post-war emergence of paralytic polio as a feared infectious disease. As cohorts aged, people who grew up with cleaner water became adults susceptible to polio; for all intents and purposes, this phenomenon did not exist before the early twentieth century.

Given the close fit of the mortality data to the hygiene hypothesis, we do not dwell on issues of statistical significance. While we have focused on the results as differences, ratios, and their graphical presentation, significance is well-documented in Appendix B. Consider ages 5–14 (table 9, p.36), and the key decade of 1940–49 — the last complete decade before the vaccine, and the "cleanest" until that point (measured by typhoid fever death rates). In

nine of these ten years, and for both sexes, polio death rates were higher for whites than nonwhites and in one year they were equal. For males, eight of the nine white-excess years were statistically significant; seven for females. In the non-significant, non-equal years (one for males and two for females), whites' polio mortality still exceeded that of nonwhites. Contrast this with typhoid fever mortality (again, table 9): it's almost the perfect inverse, race-wise. For typhoid, and looking at the same age group (5–14) and decade (1940s), nonwhites always have higher mortality (no ties). In eight years for males (seven for females), the nonwhite typhoid excess mortality is statistically significant. In one year for males (two for females), there was a nonwhite excess that was not significant, and there was one year per sex in which significance calculations were not feasible because mortality rates were zero for whites.

Conclusion

Consider again figure 1, the Casablanca antibody data of Paul and Horstmann (1955), from a time in Morocco when there was no paralytic polio except in the French expatriate community. These are cross-sectional data, but come from the period of stable equilibrium prior to the emergence of poliomyelitis in Morocco. Thus, the antibody profile by age can be considered to be a cohort (or individual) profile as well. Babies are born with protective maternal antibodies. This reflects the fact that their mothers experienced asymptomatic polio infection and were constantly boosted by exposure to the poliovirus. During infancy, at varying rates, these maternal antibodies fade. In the omnipresence of poliovirus, when maternal antibodies have declined to the point at which they provide only partial protection, it sets the stage for asymptomatic polio infection, thus generating lifelong protective immunity. Maternal antibody immunity is passive, while immunity from asymptomatic infection is adaptive (i.e., active). If this does not occur in infancy, it happens in early childhood. This generates the steeply declining — and then steeply increasing — prevalence of neutralizing antibody by age, followed by consistently high levels at older ages (figure 1).

Consider again the polio hygiene sub-hypotheses (p.6):

1. [Non-infant polio death rates increase, until 1955.] *The data support this.*

2. [Polio mortality increases greater above age 5.] *The data support this.*

3. [Nonwhites:]
   (a) [increase less than whites] *The data support this.*
   (b) [except during infancy] *The data support this.*
   (c) [decline with IPV] *The data only partly support this, as it appears that nonwhites had less access to the vaccine.*
4. [patterns similar by sex] *The data support this.*

In short, the racially cross-classified polio mortality data conform well to the hygiene hypothesis. Martinez-Bakker et al. (2015) write: "We also show that the historical emergence of epidemic polio was largely a consequence of demographic trends rather than improvements in hygiene" (p.17). We find that racial trends in poliomyelitis mortality in the United States are at odds with that conclusion.

From Polio's emergence as an epidemic disease in the northeast in 1916, through the vaccine era that began in 1955, and until its de facto elimination in the 1980s (Strebel et al., 1992), polio's epidemiology in the United States was consistent with the hygiene hypothesis. Internationally, the country-by-country emergence of paralytic polio throughout the twentieth century also reflected the global dimensions of the hygiene hypothesis: "Within the last decade or so, many countries have experienced relatively serious outbreaks of poliomyelitis for the first time" (Payne, 1955, p.393).

Changes in polio transmission resulted in an increase in the average age of acquisition, which in turn drove changes in polio epidemiology throughout the twentieth century. In this paper, we documented the twentieth-century evolution of polio mortality by sex, race and age; these patterns are best explained by the hygiene hypothesis. The birth-rate hypothesis does not include race and therefore does not account for the observed patterns. Typhoid mortality data, on the other hand, permit racially-specific versions of the hygiene hypothesis.

Rising polio mortality in the postwar era captured the imagination because it occurred even as life expectancy was growing (Berin et al., 1989) and infectious disease case fatality rates were falling, with much attribution of this to penicillin (f.e. Burnet, 1951). Mortality improvements occurred even with non-bacterial diseases such as measles (Gindler et al., 2004). With poliomyelitis, relief had to wait for the IPV vaccine in 1955. The racial mortality data show that this resulted in a racial flip-flop, with polio becoming a nonwhite-excess killer. While the pivotal postwar period in polio coincided

with the baby boom, our racially-stratified analysis confirms that the hygiene hypothesis is alive and well. Or: throw out the baby boom in favor of the bathwater.

Appendix A: Data sources

For 1910–53, we used mortality rate data, and for 1954–69, we computed mortality rates from death and population counts, using these sources:

year range	rate data	death counts	population counts
1910–53	A	—	B
1954	—	U.S. Department of Health, Education, and Welfare (1956), tab. 52A	B
1955	—	U.S. Department of Health, Education, and Welfare (1957), tab. 53	B
1956	—	U.S. Department of Health, Education, and Welfare (1958), tab. 64A	B
1957	—	U.S. Department of Health, Education, and Welfare (1959), tab. 64	B
1958	—	U.S. Department of Health, Education, and Welfare (1960), tab. 73	B
1959–67	—	National Center for Health Statistics (2006)	B, C
1968–69	—	National Center for Health Statistics (2014)	C

A=U.S. Department of Health, Education, and Welfare (1956)
B=National Center for Health Statistics (2009*a*)
C=National Center for Health Statistics (2009*b*)

The following codes in the International Classification of Diseases (ICD) correspond to the analyzed causes of death:

year range	ICD revision	ICD code	
		typhoid	polio
1914–20	2	1	63A
1921–29	3	1A	22
1930–38	4	1	16
1939–48	5	1	36
1949–57	6	040	080
1958–67	7	040	080
1968–69	8	001	040–043

Data on births (figure 9) come from the crude birth rate by race (Grove and Hetzel, 1968, p.114), multiplied by the race-specific total population size (from National Center for Health Statistics, 2009*a*,*b*).

Appendix B: Descriptive statistics and tests

Tables 1–6 (pp.28–33) give all-years and decadal arithmetic and geometric means (Schoen, 1970) of polio and typhoid death rates, broken down by age (one table per age-panel of figures 3 and 5). The figures have some gaps, and the N-missing and N-zero rows of the tables clarify the origins of these. Zero values appear as gaps because figures 3 and 5 have logarithmic scale, and therefore cannot plot zeros, whereas missing values are simply missing. For both causes and all demographic groups, 1910–13 and 1917, are missing, so N-missing is five for the decade 1910–19 and for the total time span. All other gaps in the figures are caused by zero values. Where there are a large number of zero values, the two means (geometric and arithmetic) diverge, since zeros must be excluded from the calculation of geometric means.

Tables 7–12 (pp.34–39) summarize tests of statistical significance of white vs. non-white death rates (by age groups, sex, and disease). An example of how to read these tables is as follows. In table 7 (p.34), the upper-right corner of the table summarizes the comparisons for polio, for males. The values, are 3, 6; and then (next row) 9, 28; and then (next row) 6, 0; and then 0, 3. The first row indicates that there are 3 years in which white males have statistically-significantly higher death rates than nonwhite males, and 6 years in which nonwhite males were higher (and statistically significant). The next row indicates there were 9 years in which whites were higher but not statistically significant, and 28 years in which nonwhites were higher but not statistically significant. The next row indicates that there were 6 years in which whites were higher by virtue of nonwhites having a recorded death rate of zero, and no years in which nonwhites were higher because whites had a death rate of zero. The next row shows that there were no years in which whites and nonwhites had equal (and nonzero) death rates, but 3 years in which whites and nonwhites had equal death rates because both were zero. This concludes the comparisons for polio for males. The next block is for polio, for females (1, 2 then 12, 31, then 7, 0, then 0, 2), and so on. The same comparisons, disaggregated by decade, are shown in the lower blocks of rows. Table 7 (p.34) is for infants; subsequent tables are for other age groups.

Throughout, statistical significance is defined using a 95% uncertainty interval approach. The white and nonwhite rates are significantly different if and only if the white point estimate lies outside the 95% UI for the nonwhite estimate *and vice versa*. The 95% UIs were constructed using $(D \pm 1.96\sqrt{D})/K$, in which D is the number of deaths in a given cell, and K is

the exposure (person-years at risk) in that cell (the point estimate is D/K). This is based on the Poisson variance of death counts (Brillinger, 1986; Andreasen and Simonsen, 2011). Lack of significance often reflects the small counts of polio and typhoid deaths in later years.

[*tables begin overleaf*]

Table 1: **Descriptive statistics for data in figures. Age 0.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all years								
geomean	1.936	4.280	1.495	3.132	0.622	3.344	0.645	3.581
mean	4.517	5.505	3.883	4.434	0.971	3.823	0.756	3.786
N-miss	5	5	5	5	5	5	5	5
N-zero	3	9	2	9	13	21	17	19
1910–19								
geomean	11.763	18.035	11.140	10.964	3.846	10.073	2.831	11.534
mean	22.620	20.320	19.440	13.440	3.880	11.260	2.880	13.380
N-miss	5	5	5	5	5	5	5	5
N-zero	0	0	0	0	0	0	0	0
1920–29								
geomean	5.512	7.661	4.884	9.257	1.812	9.727	1.352	7.499
mean	5.660	8.400	4.960	9.650	1.840	10.990	1.400	7.990
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	0	0	0	0
1930–39								
geomean	3.490	5.590	3.198	3.542	1.230	2.849	0.994	4.234
mean	4.100	6.460	3.470	4.340	1.260	3.560	1.080	4.800
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	0	0	0	0
1940–49								
geomean	1.950	2.398	1.636	2.479	0.252	0.819	0.215	1.425
mean	2.160	2.780	1.860	2.670	0.230	0.640	0.210	1.170
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	2	5	1	3
1950–59								
geomean	1.070	2.059	0.721	0.871	0.079	0.464	0.062	0.432
mean	1.511	2.446	1.272	0.942	0.050	0.140	0.012	0.175
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	1	4	7	8	6
1960–69								
geomean	0.119	0.327	0.083	0.333	0.061	0.654	0.066	—
mean	0.101	0.033	0.076	0.067	0.018	0.065	0.013	0.000
N-miss	0	0	0	0	0	0	0	0
N-zero	3	9	2	8	7	9	8	10

Table 2: **Descriptive statistics for data in figures. Age 1–4.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all years								
geomean	1.765	2.013	1.222	1.912	0.953	2.309	0.745	1.869
mean	3.936	3.023	3.208	2.958	1.337	4.682	1.289	4.870
N-miss	5	5	5	5	5	5	5	5
N-zero	5	5	3	6	19	13	17	11
1910–19								
geomean	8.401	8.146	7.837	8.448	6.006	18.412	5.586	19.743
mean	19.600	10.420	15.540	11.500	6.200	18.760	5.820	20.380
N-miss	5	5	5	5	5	5	5	5
N-zero	0	0	0	0	0	0	0	0
1920–29								
geomean	4.757	4.401	4.393	4.031	2.495	9.481	2.511	10.357
mean	4.980	4.510	4.580	4.220	2.550	10.060	2.580	10.590
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	0	0	0	0
1930–39								
geomean	2.925	3.702	2.324	3.421	1.392	5.159	1.275	4.605
mean	3.320	3.890	2.600	3.530	1.470	5.400	1.370	5.070
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	0	0	0	0
1940–49								
geomean	1.884	1.462	1.460	1.592	0.196	0.742	0.196	0.516
mean	2.090	1.510	1.600	1.640	0.230	0.780	0.210	0.820
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	1	1	2	0
1950–59								
geomean	0.982	1.217	0.716	0.840	0.000	0.142	0.029	0.121
mean	1.417	1.402	1.061	1.068	0.000	0.129	0.016	0.080
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	10	2	6	4
1960–69								
geomean	0.053	0.160	0.035	0.134	0.014	0.000	0.015	0.105
mean	0.043	0.103	0.032	0.062	0.003	0.000	0.001	0.033
N-miss	0	0	0	0	0	0	0	0
N-zero	5	5	3	6	8	10	9	7

Table 3: **Descriptive statistics for data in figures. Age 5–14.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all years								
geomean	1.019	0.849	0.679	0.509	0.802	2.401	1.317	2.351
mean	1.973	0.887	1.415	0.710	1.882	7.631	2.042	9.056
N-miss	5	5	5	5	5	5	5	5
N-zero	3	7	2	4	16	11	19	9
1910–19								
geomean	2.178	1.600	1.891	0.849	8.080	29.001	8.827	34.118
mean	3.940	1.780	3.200	1.460	8.180	29.240	8.960	34.380
N-miss	5	5	5	5	5	5	5	5
N-zero	0	0	0	0	0	0	0	0
1920–29								
geomean	2.093	1.100	1.640	0.927	3.990	17.603	4.286	20.944
mean	2.360	1.130	1.830	0.960	4.110	18.090	4.450	21.570
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	0	0	0	0
1930–39								
geomean	1.775	1.089	1.243	0.922	1.779	7.410	1.908	9.005
mean	1.980	1.170	1.420	0.970	1.890	8.110	2.020	9.700
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	0	0	0	0
1940–49								
geomean	2.509	0.906	1.569	0.668	0.226	0.881	0.232	0.903
mean	2.860	0.950	1.770	0.730	0.260	1.090	0.280	1.260
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	1	0	1	0
1950–59								
geomean	0.923	0.564	0.651	0.416	0.007	0.087	0.024	0.100
mean	1.645	0.677	1.134	0.489	0.001	0.041	0.002	0.065
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	8	6	9	4
1960–69								
geomean	0.031	0.169	0.021	0.040	0.006	0.041	0.006	0.042
mean	0.035	0.064	0.028	0.026	0.002	0.022	0.001	0.022
N-miss	0	0	0	0	0	0	0	0
N-zero	3	7	2	4	7	5	9	5

Table 4: **Descriptive statistics for data in figures. Age 15–24.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all years								
geomean	0.688	0.390	0.435	0.288	1.161	4.185	1.030	4.169
mean	1.091	0.424	0.663	0.284	4.624	15.107	3.327	13.008
N-miss	5	5	5	5	5	5	5	5
N-zero	4	7	4	11	12	10	14	11
1910–19								
geomean	0.961	0.521	0.550	0.849	20.259	52.739	14.808	45.545
mean	1.320	0.600	0.740	0.340	20.560	53.080	14.940	46.060
N-miss	5	5	5	5	5	5	5	5
N-zero	0	0	0	3	0	0	0	0
1920–29								
geomean	0.925	0.387	0.553	0.191	10.143	36.072	7.345	32.271
mean	1.020	0.380	0.630	0.210	10.410	36.970	7.600	33.030
N-miss	0	0	0	0	0	0	0	0
N-zero	0	1	0	0	0	0	0	0
1930–39								
geomean	0.991	0.452	0.547	0.314	3.924	15.419	2.711	12.302
mean	1.130	0.520	0.610	0.350	4.310	17.530	2.910	13.680
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	0	0	0	0
1940–49								
geomean	1.632	0.551	0.861	0.408	0.320	1.328	0.268	1.244
mean	1.840	0.620	1.020	0.450	0.420	1.910	0.310	1.660
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	0	0	1	0
1950–59								
geomean	0.921	0.412	0.713	0.360	0.019	0.145	0.016	0.175
mean	1.325	0.480	0.983	0.361	0.010	0.128	0.009	0.122
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	1	6	2	5	4
1960–69								
geomean	0.025	0.071	0.023	0.065	0.010	0.050	0.008	0.074
mean	0.028	0.031	0.031	0.022	0.004	0.010	0.002	0.024
N-miss	0	0	0	0	0	0	0	0
N-zero	4	6	4	7	6	8	8	7

Table 5: **Descriptive statistics for data in figures. Age 25–34.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all years								
geomean	0.381	0.249	0.249	0.200	1.327	3.177	0.628	2.491
mean	0.683	0.252	0.445	0.164	3.529	8.737	2.173	6.755
N-miss	5	5	5	5	5	5	5	5
N-zero	2	12	3	16	15	10	11	11
1910–19								
geomean	0.348	0.317	0.209	0.263	17.082	30.599	9.818	22.934
mean	0.540	0.240	0.320	0.220	17.500	30.960	9.940	23.380
N-miss	5	5	5	5	5	5	5	5
N-zero	0	2	0	1	0	0	0	0
1920–29								
geomean	0.336	0.210	0.195	0.186	6.493	18.466	4.599	15.359
mean	0.390	0.180	0.230	0.080	6.680	18.710	4.770	15.620
N-miss	0	0	0	0	0	0	0	0
N-zero	0	3	0	6	0	0	0	0
1930–39								
geomean	0.379	0.243	0.273	0.184	3.358	11.172	1.804	7.925
mean	0.440	0.270	0.300	0.190	3.540	11.820	1.930	8.460
N-miss	0	0	0	0	0	0	0	0
N-zero	0	1	0	1	0	0	0	0
1940–49								
geomean	0.902	0.376	0.609	0.213	0.300	1.249	0.204	0.967
mean	1.120	0.330	0.740	0.230	0.430	1.890	0.260	1.310
N-miss	0	0	0	0	0	0	0	0
N-zero	0	2	0	0	0	0	0	0
1950–59								
geomean	1.119	0.361	0.680	0.213	0.014	0.157	0.023	0.088
mean	1.469	0.430	0.989	0.266	0.006	0.132	0.017	0.044
N-miss	0	0	0	0	0	0	0	0
N-zero	0	0	0	0	6	3	5	5
1960–69								
geomean	0.042	0.087	0.023	0.108	0.011	0.079	0.010	0.063
mean	0.066	0.056	0.031	0.026	0.001	0.024	0.004	0.025
N-miss	0	0	0	0	0	0	0	0
N-zero	2	4	3	8	9	7	6	6

Table 6: **Descriptive statistics for data in figures. Age 35–44.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all years								
geomean	0.196	0.191	0.125	0.170	1.051	2.247	0.919	2.298
mean	0.301	0.123	0.173	0.116	2.811	6.648	1.671	5.006
N-miss	5	5	5	5	5	5	5	5
N-zero	2	25	2	22	13	8	17	12
1910–19								
geomean	0.270	0.387	0.172	0.311	13.195	23.448	7.459	17.573
mean	0.340	0.160	0.220	0.200	13.520	23.760	7.600	17.820
N-miss	5	5	5	5	5	5	5	5
N-zero	0	3	0	2	0	0	0	0
1920–29								
geomean	0.202	0.245	0.137	0.200	5.797	15.425	3.480	11.978
mean	0.220	0.050	0.150	0.080	6.060	15.620	3.620	12.190
N-miss	0	0	0	0	0	0	0	0
N-zero	0	8	0	6	0	0	0	0
1930–39								
geomean	0.202	0.208	0.123	0.142	2.055	6.805	1.433	5.075
mean	0.220	0.180	0.130	0.150	2.170	7.160	1.530	5.370
N-miss	0	0	0	0	0	0	0	0
N-zero	0	3	0	1	0	0	0	0
1940–49								
geomean	0.413	0.157	0.191	0.193	0.383	1.258	0.215	0.744
mean	0.490	0.140	0.230	0.160	0.440	1.730	0.240	0.940
N-miss	0	0	0	0	0	0	0	0
N-zero	0	2	0	2	0	0	0	0
1950–59								
geomean	0.381	0.191	0.233	0.147	0.036	0.124	0.010	0.129
mean	0.528	0.193	0.308	0.121	0.026	0.152	0.002	0.094
N-miss	0	0	0	0	0	0	0	0
N-zero	0	1	0	3	5	1	8	4
1960–69								
geomean	0.026	0.124	0.025	0.110	0.010	0.075	0.009	0.110
mean	0.028	0.031	0.025	0.028	0.002	0.023	0.001	0.028
N-miss	0	0	0	0	0	0	0	0
N-zero	2	8	2	8	8	7	9	8

Table 7: **Summary of tests of significance for data in figures. Age 0.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all-years								
higher (sig.)	3	6	1	2	0	12	0	12
higher (n.s.)	9	28	12	31	2	15	0	20
higher (other=0)	6	0	7	0	13	5	6	4
Equal | both=0	0	3	0	2	0	8	0	13
1910–19								
higher (sig.)	1	0	1	0	0	2	0	2
higher (n.s.)	0	4	2	2	0	3	0	3
higher (other=0)	0	0	0	0	0	0	0	0
Equal | both=0	0	0	0	0	0	0	0	0
1920–29								
higher (sig.)	0	1	0	2	0	8	0	7
higher (n.s.)	3	6	0	8	0	2	0	3
higher (other=0)	0	0	0	0	0	0	0	0
Equal | both=0	0	0	0	0	0	0	0	0
1930–39								
higher (sig.)	0	3	0	0	0	1	0	3
higher (n.s.)	3	4	2	8	2	7	0	7
higher (other=0)	0	0	0	0	0	0	0	0
Equal | both=0	0	0	0	0	0	0	0	0
1940–49								
higher (sig.)	2	0	0	0	0	1	0	0
higher (n.s.)	2	6	3	7	0	2	0	7
higher (other=0)	0	0	0	0	5	2	2	0
Equal | both=0	0	0	0	0	0	0	0	1
1950–59								
higher (sig.)	0	2	0	0	0	0	0	0
higher (n.s.)	1	7	5	4	0	1	0	0
higher (other=0)	0	0	1	0	5	2	2	4
Equal | both=0	0	0	0	0	0	2	0	4
1960–69								
higher (sig.)	0	0	0	0	0	0	0	0
higher (n.s.)	0	1	0	2	0	0	0	0
higher (other=0)	6	0	6	0	3	1	2	0
Equal | both=0	0	3	0	2	0	6	0	8

Table 8: **Summary of tests of significance for data in figures. Age 1–4.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all-years								
higher (sig.)	8	4	5	3	0	28	0	26
higher (n.s.)	13	23	10	27	0	5	0	9
higher (other=0)	1	1	4	1	3	9	2	8
Equal | both=0	1	4	3	2	0	10	1	9
1910–19								
higher (sig.)	1	0	2	1	0	5	0	5
higher (n.s.)	1	3	0	2	0	0	0	0
higher (other=0)	0	0	0	0	0	0	0	0
Equal | both=0	0	0	0	0	0	0	0	0
1920–29								
higher (sig.)	2	0	1	0	0	10	0	10
higher (n.s.)	4	4	3	3	0	0	0	0
higher (other=0)	0	0	0	0	0	0	0	0
Equal | both=0	0	0	3	0	0	0	0	0
1930–39								
higher (sig.)	1	2	0	1	0	10	0	10
higher (n.s.)	1	6	1	8	0	0	0	0
higher (other=0)	0	0	0	0	0	0	0	0
Equal | both=0	0	0	0	0	0	0	0	0
1940–49								
higher (sig.)	2	0	1	0	0	3	0	1
higher (n.s.)	5	2	3	6	0	5	0	7
higher (other=0)	0	0	0	0	1	1	0	2
Equal | both=0	1	0	0	0	0	0	0	0
1950–59								
higher (sig.)	2	2	1	1	0	0	0	0
higher (n.s.)	2	4	3	5	0	0	0	1
higher (other=0)	0	0	0	0	0	8	2	4
Equal | both=0	0	0	0	0	0	2	1	2
1960–69								
higher (sig.)	0	0	0	0	0	0	0	0
higher (n.s.)	0	4	0	3	0	0	0	1
higher (other=0)	1	1	4	1	2	0	0	2
Equal | both=0	0	4	0	2	0	8	0	7

Table 9: **Summary of tests of significance for data in figures. Age 5–14.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all-years								
higher (sig.)	27	1	25	0	0	33	0	32
higher (n.s.)	10	6	11	10	0	4	0	2
higher (other=0)	4	0	4	2	2	7	2	12
Equal | both=0	4	3	3	0	0	9	0	7
1910–19								
higher (sig.)	1	0	3	0	0	5	0	5
higher (n.s.)	1	1	1	1	0	0	0	0
higher (other=0)	0	0	0	0	0	0	0	0
Equal | both=0	2	0	0	0	0	0	0	0
1920–29								
higher (sig.)	6	0	5	0	0	10	0	10
higher (n.s.)	4	0	4	0	0	0	0	0
higher (other=0)	0	0	0	0	0	0	0	0
Equal | both=0	0	0	1	0	0	0	0	0
1930–39								
higher (sig.)	6	0	3	0	0	10	0	10
higher (n.s.)	3	0	4	2	0	0	0	0
higher (other=0)	0	0	0	0	0	0	0	0
Equal | both=0	1	0	1	0	0	0	0	0
1940–49								
higher (sig.)	8	0	7	0	0	8	0	7
higher (n.s.)	1	0	2	0	0	1	0	2
higher (other=0)	0	0	0	0	0	1	0	1
Equal | both=0	1	0	1	0	0	0	0	0
1950–59								
higher (sig.)	6	0	6	0	0	0	0	0
higher (n.s.)	1	3	0	4	0	2	0	0
higher (other=0)	0	0	0	0	0	2	1	6
Equal | both=0	0	0	0	0	0	6	0	3
1960–69								
higher (sig.)	0	1	1	0	0	0	0	0
higher (n.s.)	0	2	0	3	0	1	0	0
higher (other=0)	4	0	4	2	2	4	1	5
Equal | both=0	0	3	0	0	0	3	0	4

Table 10: **Summary of tests of significance for data in figures. Age 15–24.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all-years								
higher (sig.)	28	0	23	0	0	33	0	33
higher (n.s.)	12	5	12	4	0	5	0	3
higher (other=0)	4	1	7	0	5	7	5	8
Equal | both=0	2	3	5	4	0	5	0	6
1910–19								
higher (sig.)	2	0	1	0	0	5	0	5
higher (n.s.)	2	1	0	1	0	0	0	0
higher (other=0)	0	0	3	0	0	0	0	0
Equal | both=0	0	0	0	0	0	0	0	0
1920–29								
higher (sig.)	6	0	6	0	0	10	0	10
higher (n.s.)	2	0	2	0	0	0	0	0
higher (other=0)	1	0	0	0	0	0	0	0
Equal | both=0	1	0	2	0	0	0	0	0
1930–39								
higher (sig.)	5	0	4	0	0	10	0	10
higher (n.s.)	5	0	4	0	0	0	0	0
higher (other=0)	0	0	0	0	0	0	0	0
Equal | both=0	0	0	2	0	0	0	0	0
1940–49								
higher (sig.)	9	0	6	0	0	8	0	8
higher (n.s.)	0	0	3	0	0	2	0	1
higher (other=0)	0	0	0	0	0	0	0	1
Equal | both=0	1	0	1	0	0	0	0	0
1950–59								
higher (sig.)	6	0	6	0	0	0	0	0
higher (n.s.)	2	2	2	1	0	2	0	1
higher (other=0)	0	0	1	0	2	6	4	5
Equal | both=0	0	0	0	0	0	0	0	0
1960–69								
higher (sig.)	0	0	0	0	0	0	0	0
higher (n.s.)	1	2	1	2	0	1	0	1
higher (other=0)	3	1	3	0	3	1	1	2
Equal | both=0	0	3	0	4	0	5	0	6

Table 11: **Summary of tests of significance for data in figures. Age 25–34.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all-years								
higher (sig.)	17	0	17	0	0	31	0	33
higher (n.s.)	17	7	9	9	0	7	0	5
higher (other=0)	11	1	13	0	2	7	5	5
Equal | both=0	1	1	4	3	0	8	1	6
1910–19								
higher (sig.)	0	0	1	0	0	5	0	5
higher (n.s.)	2	0	0	2	0	0	0	0
higher (other=0)	2	0	1	0	0	0	0	0
Equal | both=0	1	0	1	0	0	0	0	0
1920–29								
higher (sig.)	2	0	0	0	0	10	0	10
higher (n.s.)	4	1	2	2	0	0	0	0
higher (other=0)	3	0	6	0	0	0	0	0
Equal | both=0	0	0	0	0	0	0	0	0
1930–39								
higher (sig.)	3	0	3	0	0	10	0	10
higher (n.s.)	4	2	3	2	0	0	0	0
higher (other=0)	1	0	1	0	0	0	0	0
Equal | both=0	0	0	1	0	0	0	0	0
1940–49								
higher (sig.)	5	0	6	0	0	6	0	8
higher (n.s.)	3	0	2	0	0	4	0	2
higher (other=0)	2	0	0	0	0	0	0	0
Equal | both=0	0	0	2	0	0	0	0	0
1950–59								
higher (sig.)	6	0	7	0	0	0	0	0
higher (n.s.)	3	1	2	1	0	3	0	2
higher (other=0)	0	0	0	0	1	4	2	2
Equal | both=0	0	0	0	0	0	2	1	3
1960–69								
higher (sig.)	1	0	0	0	0	0	0	0
higher (n.s.)	1	3	0	2	0	0	0	1
higher (other=0)	3	1	5	0	1	3	3	3
Equal | both=0	0	1	0	3	0	6	0	3

Table 12: **Summary of tests of significance for data in figures. Age 35–44.**

	Polio				Typhoid			
	Males		Females		Males		Females	
	White	Non-wh.	White	Non-wh.	White	Non-wh.	White	Non-wh.
all-years								
higher (sig.)	9	0	3	0	0	33	0	31
higher (n.s.)	6	11	10	13	0	5	0	5
higher (other=0)	23	0	20	0	2	7	2	7
Equal | both=0	4	2	7	2	2	6	0	10
1910–19								
higher (sig.)	0	0	0	0	0	4	0	4
higher (n.s.)	1	1	0	3	0	1	0	1
higher (other=0)	3	0	2	0	0	0	0	0
Equal | both=0	0	0	0	0	0	0	0	0
1920–29								
higher (sig.)	0	0	0	0	0	10	0	10
higher (n.s.)	0	2	1	2	0	0	0	0
higher (other=0)	8	0	6	0	0	0	0	0
Equal | both=0	0	0	1	0	0	0	0	0
1930–39								
higher (sig.)	0	0	0	0	0	10	0	10
higher (n.s.)	2	3	2	3	0	0	0	0
higher (other=0)	3	0	1	0	0	0	0	0
Equal | both=0	2	0	4	0	0	0	0	0
1940–49								
higher (sig.)	4	0	1	0	0	8	0	7
higher (n.s.)	2	0	2	3	0	1	0	3
higher (other=0)	2	0	2	0	0	0	0	0
Equal | both=0	2	0	2	0	1	0	0	0
1950–59								
higher (sig.)	5	0	2	0	0	1	0	0
higher (n.s.)	1	3	5	0	0	3	0	1
higher (other=0)	1	0	3	0	0	4	1	5
Equal | both=0	0	0	0	0	1	1	0	3
1960–69								
higher (sig.)	0	0	0	0	0	0	0	0
higher (n.s.)	0	2	0	2	0	0	0	0
higher (other=0)	6	0	6	0	2	3	1	2
Equal | both=0	0	2	0	2	0	5	0	7

Appendix C: Racial comparisons for other diseases

To illustrate how unusual it is to see a nonwhite excess in infectious disease mortality — as in figure 6, for poliomyelitis — figure 11 (overleaf) shows white/nonwhite comparisons for a battery of other infectious diseases. Because twelve diseases are compared in one graph, age groups are collapsed using the age-adjusted death rate. Polio and typhoid are shown for comparison (these two diseases are repeated from figures 4 and 6). None of the earlier graphs show the age-adjusted death rate — only the component (age-specific) death rates, and only up to age 45. The ten other infectious diseases are: dysentery; meningococcal infections; GCDE (gastritis, colitis, duodenitis, and enteritis); TB (tuberculosis, all forms); pertussis; measles; diphtheria; PI (pneumonia and influenza); RF (rheumatic fever), and syphilis. The data come from U.S. Department of Health, Education, and Welfare (1956), and this figure includes all infectious diseases in that source. The typhoid series ends in 1948 because the white age-adjusted death rate is zero in all later data, meaning the ratio cannot be calculated.

All diseases in figure 11 follow the typhoid pattern of nonwhite excess, not the polio pattern — except meningococcal disease, measles, and diphtheria. None of these three share polio's pattern of declining ratio during the 1940s and early 1950s. Meningococcal infections and measles show a lot of noise around the line of equality, and moreover from 1935 onward show nonwhite excess for the preponderance of years. Diphtheria, on the other hand, trends clearly toward greater and greater nonwhite excess after 1940. The point here is that the polio pattern by race is unlike that of any other infectious disease, even those which are exceptions to the rule that nonwhite death rates always exceeds that of whites.

[*figure is overleaf*]

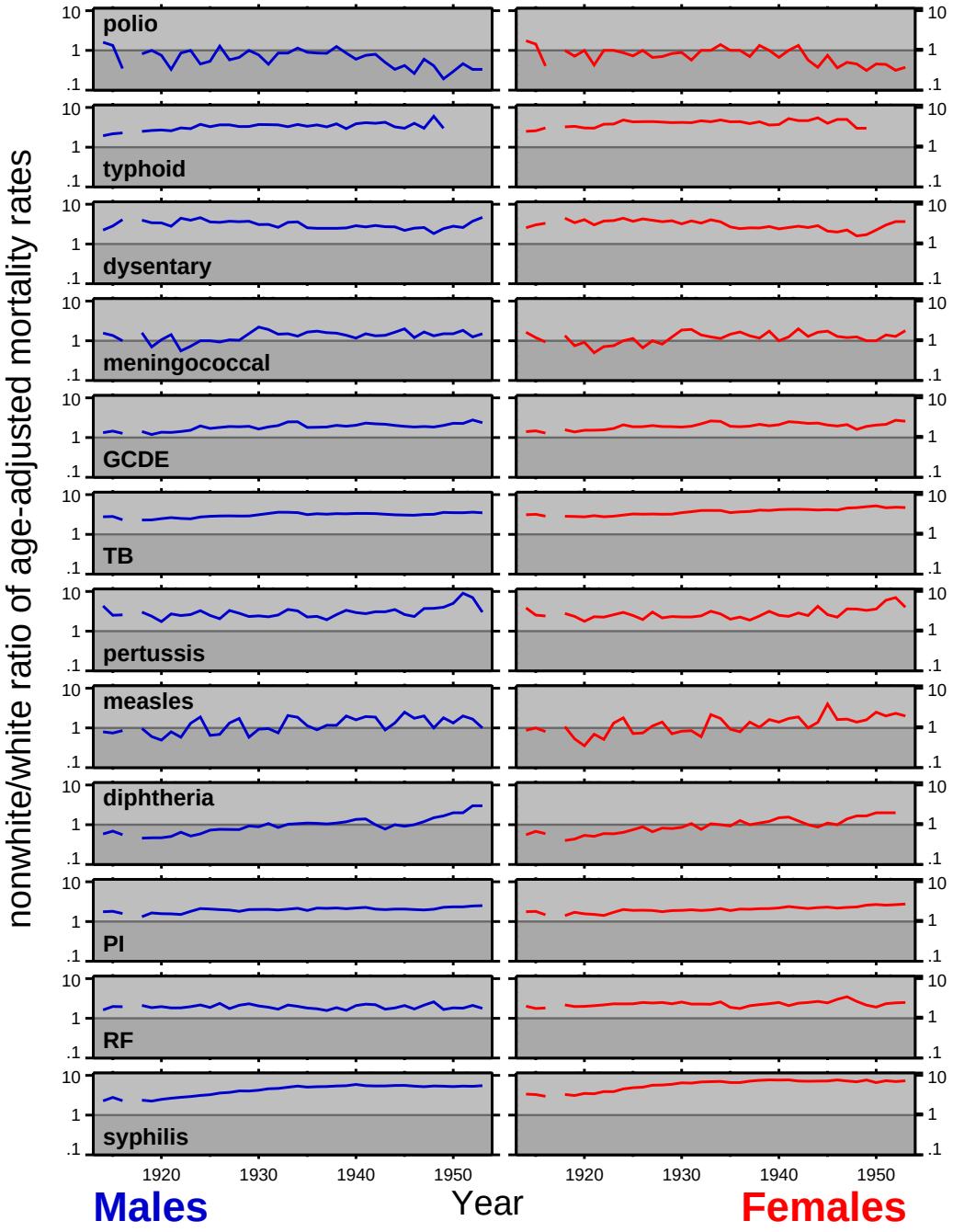

Figure 11: Time series of nonwhite/white ratio of age-adjusted death rates, 1914–53. Polio; typhoid; dysentery; meningococcal infections; GCDE (gastritis, colitis, duodenitis, and enteritis); TB (tuberculosis, all forms); pertussis; measles; diphtheria; PI (pneumonia and influenza); RF (rheumatic fever); and syphilis.

Appendix D: Heatmaps

These two figures present the same data as figures 3 and 5, but as age×time heatmaps. These do not permit white:nonwhite comparisons on the same axes, as figures 3 and 5 do, but have the advantage of not segregating the age groups into discrete panels. These heatmaps show clearly that typhoid death rates decline continuously throughout the period, while polio death rates rise for whites in the 1940s and 1950s, particularly at older age groups. The color scale is inverse hyperbolic sign-transformed, resulting in no missing values for zeros; data for 1917 are missing in the original source. The maximum values for polio occurred in 1916, associated with the huge epidemic in the northeast (Nicoll, 1917; Lavinder et al., 1918). Heatmaps for both diseases are constructed using the same maximal possible value: 83.6 per 100,000 (corresponding to polio in 1916, for white male infants). The maximum observed value for typhoid is lower, 59.6/100,000 (for 1918, nonwhite males).

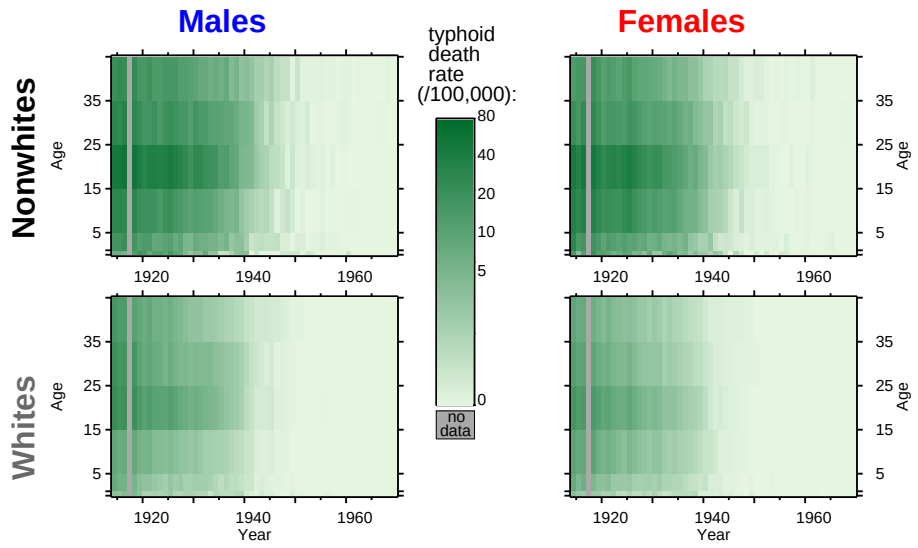

Figure 12: Heatmap of typhoid fever death rates over time. Compare to figure 3.

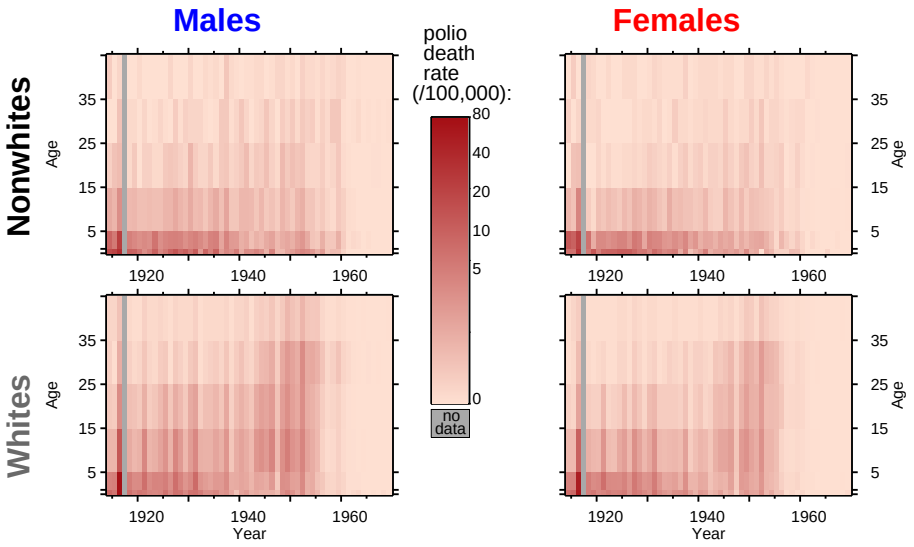

Figure 13: Heatmap of poliomyelitis death rates over time. Compare to figure 5.

Works Cited

Alsan, Marcella and Claudia Goldin. 2018. "Watersheds in child mortality: The role of effective water and sewerage infrastructure, 1880 to 1920." NBER Working Paper 21263, National Bureau of Economic Research. DOI: 10.3386/w21263.

Anderson, Roy M. and Robert M. May. 1991. *Infectious diseases of humans: Dynamics and control*. Oxford University Press.

Andreasen, Viggo and Lone Simonsen. 2011. "The perils of using annual all-cause mortality data to estimate pandemic influenza burden." *Vaccine* 29:B49–B55. DOI: 10.1016/j.vaccine.2011.03.061.

Asturias, Edwin J., Erica L. Dueger, Saad B. Omer, Arturo Melville, Silvia V. Nates, Majid Laassri, Konstantin Chumakov, and Neal A. Halsey. 2007. "Randomized trial of inactivated and live polio vaccine schedules in Guatemalan infants." *Journal of Infectious Diseases* 196(5):692–698. DOI: 10.1086/520546.

Aycock, W. Lloyd and Gilcin P. Meadors. 1948. "The principles of variolation as exemplified by sub clinical immunization in poliomyelitis in cooler and warmer climates." *American Journal of the Medical Sciences* 215(3):296–310.

Bartlett, M. S. 1957. "Measles periodicity and community size (with discsussion)." *Journal of the Royal Statistical Society, Series A* 120(1):48–70. DOI: 10.2307/2342553.

Beach, Brian, Joseph Ferrie, Martin Saavedra, and Werner Troesken. 2016. "Typhoid fever, water quality, and human capital formation." *Journal of Economic History* 76(1):41–75. DOI: 10.1017/S0022050716000413.

Berin, Bamet N., George J. Stolnitz, and Aaron Tenebein. 1989. "Mortality trends

of males and females over the ages." *Transactions of the Society of Actuaries* 41:1–19.

Brillinger, David R. 1986. "The natural variability of vital rates and associated statistics." *Biometrics* 42(4):693–734. DOI: 10.2307/2530689.

Bunimovich-Mendrazitsky, Svetlana and Lewi Stone. 2005. "Modeling polio as a disease of development." *Journal of Theoretical Biology* 237(3):302–315. DOI: 10.1016/j.jtbi.2005.04.017.

Burbidge, John B., Lonnie Magee, and A. Leslie Robb. 1988. "Alternative transformations to handle extreme values of the dependent variable." *Journal of the American Statistical Association* 83(401):123–127. DOI: 10.1080/01621459.1988.10478575.

Burnet, F. M. 1951. "Viruses." *Scientific American* 184(5):43–51. DOI: 10.1038/scientificamerican0551-43.

Candioti, Carlos M. Vera, Oswaldo P. Campos, Min Sein, Ernest Couture, Hernan Romero, Juan Ruiz-Mora, Fernando Lopez Fernandez, J. A. Slavik, Henry C. H. Lassen, Luis F. Thomen, W. H. Bradley, Leo Arturo Kaprio, Pierre R. LePine, A. Codounis, Carlos M. Monson-Malice, Bjarni Jonsson, V. R. Thaymanaswami, Hashim Al Witry, Juan Farill, S. Van Creveld, Duncan Cook, M. Berendson, Gunnar Orn, Hans Zellweger, Fevzi Gunalp, B. A. Dorimer, Enrique M. Claveaux, and Rolla E. Dyer. 1949. "Poliomyelitis throughout the world." In Fishbein (1949), pp. 327–353.

Ciocco, Antonio. 1940*a*. "Sex differences in morbidity and mortality." *Quarterly Review of Biology* 15(1):59–73. DOI: 10.1086/394601.

———. 1940*b*. "Sex differences in morbidity and mortality (concluded)." *Quarterly Review of Biology* 15(2):192–210. DOI: 10.1086/394606.

Cliff, Andrew D., Peter Haggett, and Donna F. Stroup. 1992*a*. "The geographic structure of measles epidemics in the northeastern United States." *American Journal of Epidemiology* 136(5):592–602. DOI: 10.1093/oxfordjournals.aje.a116537.

Cliff, Andrew D., Peter Haggett, Donna F. Stroup, and Elizabeth Cheney. 1992*b*. "The changing geographical coherence of measles morbidity in the United States, 1962–88." *Statistics in Medicine* 11(11):1409–1424. DOI: 10.1002/sim.4780111102.

Collins, Selwyn D. 1946. "The incidence of poliomyelitis and its crippling effects, as recorded in family surveys." *Public Health Reports* 61(10):327–355. DOI: 10.2307/4585588.

Collins, Selwyn D. and Clara Councell. 1943. "Extent of immunization and case histories for diphtheria, smallpox, scarlet fever, and typhoid fever in 200,000 surveyed families in 28 large cities." *Public Health Reports* 58(30):1121–1151. DOI: 10.2307/4584540.

Condran, Gretchen A. and Eileen Crimmins-Gardner. 1978. "Public health measures and mortality in U.S. cities in the late nineteenth century." *Human Ecology* 6(1):27–54. DOI: 10.1007/BF00888565.

Condran, Gretchen A. and Harold R. Lentzner. 2004. "Early death: Mortality among young children in New York, Chicago, and New Orleans." *Journal of Interdisciplinary History* 34(3):315–354. DOI: 10.1162/002219504771997881.

Cowhig, James D. and Calvin L. Beale. 1964*a*. "Relative socioeconomic status of Southern whites and nonwhites, 1950 and 1960." *Southwestern Social Science Quarterly* 45(2):113–124. DOI: 10.2307/42867119.

———. 1964*b*. "Socioeconomic differences between white and nonwhite farm populations of the South." *Social Forces* 42(3):354–362. DOI: 10.2307/2575540.

Cutler, David and Grant Miller. 2005. "The role of public health improvements in health advances: The twentieth-century United States." *Demography* 42(1):1–22. DOI: 10.1353/dem.2005.0002.

Dandoy, Suzanne. 1967. "Measles epidemiology and vaccine use in Los Angeles County, 1963 and 1966." *Public Health Reports* 82(8):659–666. DOI: 10.2307/4593099.

Dömök, István. 1985. "Enterovirus infections: Poliomyelitis." In Derek Robinson (ed.), *Epidemiology and the community control of disease in warm climate countries*, chap. 23, pp. 306–326. Churchill Livingstone, Edinburgh, second ed.

Easterlin, Richard A. 1961. "The American baby boom in historical perspective." *American Economic Review* 51(5):869–911.

Eiben, Robert M. 1997. "The polio experience and the twilight of the contagious disease hospital." In Thomas M. Daniel and Frederick C. Robbins (eds.), *Polio*, chap. 6, pp. 97–119. University of Rochester Press, Rochester, NY.

Ewbank, Douglas C. 1987. "History of black mortality and health before 1940." *Milbank Quarterly* 65, Suppl. 1(1):100–128. DOI: 10.2307/3349953.

Ferrie, Joseph P. and Werner Troesken. 2008. "Water and Chicago's mortality transition, 1850–1925." *Explorations in Economic History* 45(1):1–16. DOI: 10.1016/j.eeh.2007.06.001.

Fishbein, Morris (ed.). 1949. *Poliomyelits: Papers and discussions presented at the First International Poliomyelitis Conference, New York, 1948*. J. B. Lippincott, Philadelphia.

Francis, Thomas and Robert F. Korns. 1955. "Evaluation of 1954 field trial of poliomyelitis vaccine: Synopsis of summary report." *American Journal of the Medical Sciences* 229(6):603–612. DOI: 10.1097/00000441-195506000-00001.

Francis, Thomas, Jr. 1955. "An evaluation of the 1954 poliomyelitis vaccine trials: Summary report." *American Journal of Public Health* 45(5, pt.2):1–50.

Frost, W. H. 1916. "Relationship of milk supplies to typhoid fever." *Public Health Reports* 31(48):3291–3302. DOI: 10.2307/4574300.

Gilliam, Alexander G., Fay M. Hemphill, and Jean H. Gerende. 1949*a*. "Average poliomyelitis incidence reported in the counties of the United States, 1932–1946." *Public Health Reports* 64(49):1575–1584.

———. 1949*b*. "Poliomyelitis epidemic recurrence in the counties of the United States, 1932–1946." *Public Health Reports* 64(49):1584–1595. DOI: 10.2307/4587175.

Gindler, Jacqueline, Sarah Tinker, Lauri Markowitz, William Atkinson, Loring Dales, and Mark Papania. 2004. "Acute measles mortality in the United States, 1987–2002." *Journal of Infectious Diseases* 189(Suppl. 1):S69–S77. DOI: 10.1086/378565.

Goldblum, Natan and Joseph L. Melnick. 1952. "Complement-fixing antibodies to type 2 (Lansing) poliomyelitis virus in a normal population of a subtropical area." *Journal of Experimental Medicine* 96(2):175–185. DOI: 10.1084/jem.96.2.175.

Greene, William H. 1997. *Econometric analysis*. Prentice Hall, Upper Saddle River, NJ, 3rd ed.

Grenfell, B. T. and R. M. Anderson. 1985. "The estimation of age-related rates of infection from case notifications and sero-

logical data." *Journal of Hygiene* 95(2):419–436. DOI: 10.1017/S0022172400062859.

Gröschel, Dieter H. M. and Richard B. Hornick. 1981. "Who introduced typhoid vaccination: Almroth Wright or Richard Pfeiffer?" *Reviews of Infectious Diseases* 3(6):1251–1254. DOI: 10.1093/clinids/3.6.1251.

Grove, Robert D. and Alice M. Hetzel. 1968. *Vital statistics rates in the United States, 1940–1960*. United States Department of Health, Education, and Welfare: National Center for Health Statistics, Washington DC.

Hammon, William McD. and Ernest H. Ludwig. 1957. "Possible protective effect of previous type 2 infection against paralytic poliomyelitis due to type 1 virus." *American Journal of Hygiene* 66(3):274–280. DOI: 10.1093/oxfordjournals.aje.a119900.

Hanna-Attisha, Mona, Jenny LaChance, Richard Casey Sadler, and Allison Champney Schnepp. 2016. "Elevated blood lead levels in children associated with the Flint drinking water crisis: A spatial analysis of risk and public health response." *American Journal of Public Health* 106(2):283–290. DOI: 10.2105/AJPH.2015.303003.

Healy, Kieran. 2018. "Visualizing the baby boom." *Socius* 4:2378023118777,324. DOI: 10.1177/2378023118777324.

Hetzel, Alice M. 1997. "U.S. vital statistics system: Major activities and developments, 1950–95." DHHS Publication No. (PHS) 97-1003, National Center for Health Statistics, Hyattsville, MD.

Higgs, Robert and David Booth. 1979. "Mortality differentials within large American cities in 1890." *Human Ecology* 7(4):353–370. DOI: 10.1007/BF00888102.

Hobcraft, John, Jane Menken, and Samuel Preston. 1982. "Age, period, and cohort effects in demography: A review." *Population Index* 48(1):4–43. DOI: 10.2307/2736356.

Honig, Edward I., Joseph L. Melnick, Peter Isacson, Robert Parr, Ira L. Myers, and Mary Walton. 1956. "An endemiological study of enteric virus infections." *Journal of Experimental Medicine* 103(2):247–262. DOI: 10.1084/jem.103.2.247.

Hornick, Richard B. 1992. "Typhoid fever." In Sherwood L. Gorbach, John G. Bartlett, and Neil R. Blacklow (eds.), *Infectious diseases*, chap. 77, pp. 585–589. W. B. Saunders, Philadelphia.

Horstmann, Dorothy M. 1953. "The epidemiology and pathogenesis of poliomyelitis." *Bulletin of the New York Academy of Medicine* 29(12):910–929.

———. 1955. "Poliomyelitis: Severity and type of disease in different age groups." *Annals of the New York Academy of Sciences* 61(4):956–967. DOI: 10.1111/j.1749-6632.1955.tb42554.x.

———. 1963. "Epidemiology of poliomyelitis and allied diseases—1963." *Yale Journal of Biology and Medicine* 36(1):5–26.

Horstmann, Dorothy M., Lisbeth M. Kraft, Sarah Melnick, and Anne Mascola. 1955. "Poliomyelitis and other antibody patterns in natives of Tahiti and Raiatea." *Journal of Immunology* 75(3):249–258.

Isacson, Peter, Joseph L. Melnick, Mary Walton, Edward M. Opton, Warren Cardwell, Richard Prindle, Robert L. Parr, Ira L. Myers, and Winifred M. Mendez. 1957. "Environmental studies op endemic enteric virus infections: II. Poliovirus infections in household units." *American Journal of Hygiene* 65(1):29–42. DOI: 10.1093/oxfordjournals.aje.a119854.

Kessel, John F., Victor J. Cabasso, and Max R. Stebbins. 1956. "Poliomyelitis antibodies in inhabitants of the Society Islands, French Oceania." *Proceedings of the Society for Experimental Biology and Medicine* 91(1):132–135. DOI: 10.3181/00379727-91-22189.

Kirk, Dudley. 1996. "Demographic transition theory." *Population Studies* 50(3):361–387. DOI: 10.1080/0032472031000149536.

Kurland, Brenda F., Laura L. Johnson, Brian L. Egleston, and Paula H. Diehr. 2009. "Longitudinal data with follow-up truncated by death: Match the analysis method to research aims." *Statistical Science* 24(2):211–222. DOI: 10.1214/09-STS293.

Lavinder, Claude Hervey, Allen Weir Freeman, and Wade Hampton Frost. 1918. *Epidemiologic studies of poliomyelitis in New York City and the northeastern United States during the year 1916*. No. 91 in Public Health Bulletin, United States Public Health Service, Washington DC.

Le Bouvier, George L. 1957. "On the rise and decline of poliovirus antibodies in different human populations." *American Journal of Hygiene* 66(3):342–362. DOI: 10.1093/oxfordjournals.aje.a119907.

Leeuwenburg, J., A. G. Ferguson, and Omondi-Odhiambo. 1979. "Machakos Project studies: XIII. Spatial contagion in measles epidemics." *Tropical and Geographical Medicine* 31(2):311–320.

Lerner, William (ed.). 1975. *Historical Statistics of the United States: Colonial times to 1970*. U.S. Bureau of the Census, Washington, DC.

Levine, William C. and Paul A. Blake. 1992. "Typhoid fever." In John M. Last and Robert B. Wallace (eds.), *Maxcy-Rosenau-Last public health & preventive medicine*, pp. 173–174. Appleton & Lange, Norwalk, Connecticut, thirteenth ed.

Maldonado, Yvonne A., Victor Peña-Cruz, Maria de la Luz Sanchez, Linda Logan, Stewart Blandón, Michael F. Cantwell, Suzanne M. Matsui, Francisco Millan-Velasco, Jose Luis Valdespino, and Jaime Sepulveda. 1997. "Host and viral factors affecting the decreased immunogenicity of Sabin type 3 vaccine after administration of trivalent oral polio vaccine to rural Mayan children." *Journal of Infectious Diseases* 175(3):545–553. DOI: 10.1093/infdis/175.3.545.

Martinez-Bakker, Micaela, Aaron A. King, and Pejman Rohani. 2015. "Unraveling the transmission ecology of polio." *PLoS Biology* 13(6):1–21. DOI: 10.1371/journal.pbio.1002172.

May, Jacques M. 1950. "Map of the world distribution of poliomyelitis." *Geographical Review* 40(4):646–648. DOI: 10.2307/211108.

McCaw, Walter D. 1919. "Typhoid vaccination no substitute for sanitary precautions." *Public Health Reports* 34(13):605–622.

Meeker, Edward. 1971. "The improving health of the United States, 1850–1915." *Explorations in Economic History* 9:353–373. DOI: 10.1016/0014-4983(71)90066-0.

Mekhaiel, David N. A., Claudio T. Daniel-Ribeiro, Philip J. Cooper, and Richard J. Pleass. 2011. "Do regulatory antibodies offer an alternative mechanism to explain the hygiene hypothesis?" *Trends in Parasitology* 27(12):523–529. DOI: 10.1016/j.pt.2011.08.003.

Melnick, J. L. 1993. "Lessons from poliovirus control: Strategies for eradication." In Edouard Kurstak (ed.), *Measles and poliomyelitis: Vaccines, immunization, and control*, chap. 14, pp. 189–204. Springer, Wien. DOI: 10.1007/978-3-7091-9278-8_15.

Melnick, Joseph L. 1959. "Studies on the serological epidemiology of poliomyelitis as an index of immunity in certain Caribbean islands, British Guiana, and Ecuador." *West Indian Medical Journal* 8(4):275–298.

Melnick, Joseph L., Matilda Benyesh-Melnick, Ramiro Peña, and Martha Yow. 1961. "Effectiveness of Salk vaccine: Analysis of virologically confirmed cases of paralytic and nonparalytic poliomyelitis." *Journal of the American Medical Association* 175(13):1159–1162. DOI: 10.1001/jama.1961.03040130043010.

Melnick, Joseph L. and Nada Ledinko. 1951. "Social serology: Antibody levels in a normal young population during an epidemic of poliomyelitis." *American Journal of Hygiene* 54(3):354–382. DOI: 10.1093/oxfordjournals.aje.a119492.

———. 1953. "Development of neutralizing antibodies against the three types of poliomyelitis virus during an epidemic period: The ratio of inapparent infection to clinical poliomyelitis,." *American Journal of Hygiene* 58(2):207–222. DOI: 10.1093/oxfordjournals.aje.a119602.

Melnick, Joseph L. and Wade P. Parks. 1964. "Evaluation of community vaccination against poliomyelitis in Houston, Texas, 1962." *American Journal of Hygiene* 80(2):157–174. DOI: 10.1093/oxfordjournals.aje.a120465.

Melnick, Joseph L., John R. Paul, and Mary Walton. 1955. "Serologic epidemiology of poliomyelitis." *American Journal of Public Health* 45(4):429–437. DOI: 10.2105/AJPH.45.4.429.

Melnick, Joseph L., Manuel Ramos-Alvarez, Francis L. Black, Anthony J. Girardi, and Daizo Nagaki. 1954. "Poliomyelitis viruses in tissue culture." *Yale Journal of Biology and Medicine* 26(6):465–485.

Melnick, Joseph L., Verle Rennick, and Maria G. Molina Lozano. 1962. "Serologic epidemiology of poliomyelitis in Surinam." *Archives of Environmental Health* 4(2):202–214. DOI: 10.1080/00039896.1962.10663145.

Melnick, Joseph L., Mary Walton, Peter Isacson, Warren Cardwell, Edward M. Opton, Richard Prindle, Robert L. Parr, Ira L. Myers, and Winifred M. Mendez. 1957. "Environmental studies of endemic enteric virus infections: I. Community seroimmune patterns and poliovirus infection rates." *American Journal of Hygiene* 65(1):1–28. DOI: 10.1093/oxfordjournals.aje.a119852.

Murray, G. D. and A. D. Cliff. 1977. "A stochastic model for measles epidemics in a multi-region setting." *Transactions of the Institute of British Geographers* 2(2):158–174. DOI: 10.2307/621855.

Nathanson, C. A. 1984. "Sex differences in mortality." *Annual Review of Sociology* 10:191–213. DOI: 10.1146/annurev.so.10.080184.001203.

Nathanson, Neal and Olen M. Kew. 2010. "From emergence to eradication: The epidemiology of poliomyelitis deconstructed." *American Journal of Epidemiology* 172(11):1213–1229. DOI: 10.1093/aje/kwq320.

Nathanson, Neal and John R. Martin. 1979. "The epidemiology of poliomyelitis: Enigmas surrounding its appearance, epidemicity, and disappearance." *American Journal of Epidemiology* 110(6):672–692. DOI: 10.1093/oxfordjournals.aje.a112848.

Nathanson, Neal, Kathleen A. McGann, John Wilesmith, Ronald C. Desrosiers, and Ronald Brookmeyer. 1993. "The evolution of virus diseases: Their emergence, epidemicity, and control." *Virus Research* 29(1):3–20. DOI: 10.1016/0168-1702(93)90122-4.

National Center for Health Statistics. 2006. *Mortality multiple cause-of-death data files*. National Center for Health Statistics. http://www.nber.org/data/vital-statistics-mortality-data-multiple-cause-of-death.html. Accessed 15 January 2015.

———. 2009*a*. *Population by age groups, race, and sex for the Death Registration States, 1900–32, and for the United States, 1933–59*. Centers for Disease Control and Prevention, Hyattsville, MD. http://www.cdc.gov/nchs/data/dvs/pop0059.pdf.

———. 2009*b*. *Population by age groups, race, and sex for 1960–97*. Centers for Disease Control and Prevention, Hyattsville, MD. http://www.cdc.gov/nchs/data/dvs/pop6097.pdf.

———. 2014. *Mortality multiple cause-of-death data files*. National Center for Health Statistics. http://www.cdc.gov/nchs/nvss/mortality_public_use_data.htm Accessed 15 January 2015.

Nicoll, Matthias, Jr. 1917. "Epidemiologic data in the poliomyelitis epidemic in New York State." *American Journal of Diseases of Children* 14(2):69–79. DOI: 10.1001/archpedi.1917.01910080002001.

Oshinsky, David M. 2005. *Polio: An American story*. Oxford University Press, New York.

Parry, Christopher M., Tran Tinh Hien, Gordon Dougan, Nicholas J. White, and Jeremy J. Farrar. 2002. "Typhoid fever." *New England Journal of Medicine* 347(22):1770–1782. DOI: 10.1056/NEJMra020201.

Parsons, C. G. 1948. "Penicillin and sulphonamides in typhoid fever: Experience of physicians in military hospitals in the Middle East." *Lancet* 251(6501):510–513. DOI: 10.1016/S0140-6736(48)90723-5.

Paul, John R. 1952. "Present concepts and recent advances in acute poliomyelitis." *Archives of Internal Medicine* 90(2):271–279. DOI: 10.1001/archinte.1952.00240080137014.

———. 1958. "Endemic and epidemic trends of poliomyelitis in Central and South America." *Bulletin of the World Health Organization* 19(4):747–758.

———. 1971. *A history of poliomyelitis*. Yale University Press, New Haven.

Paul, John R. and Dorothy M. Horstmann. 1955. "A survey of poliomyelitis virus antibodies in French Morocco." *American Journal of Tropical Medicine and Hygiene* 4(3):512–524. DOI: 10.4269/ajtmh.1955.4.512.

Paul, John R., Joseph L. Melnick, VoHammie H. Barnett, and Natan Goldblum. 1952*a*. "A survey of neutralizing antibodies to poliomyelitis virus in Cairo, Egypt." *American Journal of Hygiene* 55(3):402–413.

Paul, John R., Joseph L. Melnick, and John T. Riordan. 1952*b*. "Comparative neutralizing antibody patterns to Lansing (type 2) poliomyelitis virus in different populations." *American Journal of Hygiene* 56(3):232–251. DOI: 10.1093/oxfordjournals.aje.a119549.

Payne, A. M.-M. 1955. "Poliomyelitis as a world problem." In Morris Fishbein (ed.), *Poliomyelits: Papers and discussions presented at the Third International Poliomyelitis Conference, Rome, 1954*, chap. 393-400. J. B. Lippincott, Philadelphia.

Pfeiffer, Julie K. 2010. "Innate host barriers to viral trafficking and population diversity: Lessons learned from poliovirus." *Advances in Virus Research* 77:85–118. DOI: 10.1016/B978-0-12-385034-8.00004-1.

Preston, Samuel H., Irma T. Elo, Mark E. Hill, and Ira Rosenwaike. 2003. *The demography of African Americans 1930–1990*. Kluwer, Dordrecht, Netherlands. DOI: 10.1007/978-94-017-0325-3.

Project Tycho. 2016. http://tycho.pitt.edu/. Accessed 17 November 2016.

Pyle, Gerald F. 1973. "Measles as an urban health problem: The Akron example."

Economic Geography 49(4):344–356. DOI: 10.2307/143237.

Rhodes, A. J. 1955. "Poliomyelitis and the school medical health officer." *Canadian Journal of Public Health* 46(9):355–362.

Rivers, Thomas M. 1948. "Certain public-health aspects of infectious diseases." *New England Journal of Medicine* 238(2):37–44. DOI: 10.1056/NEJM194801082380201.

Rogers, Naomi. 1992. *Dirt and disease: Polio before FDR*. Rutgers University Press, New Brunswick.

———. 2014. *Polio wars: Sister Elizabeth Kenny and the golden age of American medicine*. Oxford University Press, New York.

Rosenberg, Charles E. 1962. *The cholera years: The United States in 1832, 1849, and 1866*. University of Chicago Press.

Ryder, Norman B. 1965. "The cohort as a concept in the study of social change." *American Sociological Review* 30(6):843–861.

Sabin, Albert B. 1947. "The epidemiology of poliomyelitis: Problems at home and among the armed forces abroad." *Journal of the American Medical Association* 134(9):749–756. DOI: 10.1001/jama.1947.02880260007003.

———. 1949. "Epidemiologic patterns of poliomyelitis in different parts of the world." In Fishbein (1949), pp. 3–33.

Schoen, Robert. 1970. "The geometric mean of the age-specific death rates as a summary index of mortality." *Demography* 7(3):317–324. DOI: 10.2307/2060150.

Schoub, Barry D., Sylvia Johnson, Jo McAnerney, Linda Gilbertson, Katalina I. M. Klaassen, and Stephanus G. Reinach. 1988. "Monovalent neonatal polio immunization: A strategy for the developing world." *Journal of Infectious Diseases* 157(4):836–839. DOI: 10.1093/infdis/157.4.836.

Sheps, Mindel C. 1958. "Shall we count the living or the dead?" *New England Journal of Medicine* 259(25):1210–1214. DOI: 10.1056/NEJM195812182592505.

———. 1959. "An examination of some methods of comparing several rates or proportions." *Biometrics* 15(1):87–97. DOI: 10.2307/2527603.

Strebel, Peter M., Roland W. Sutter, Stephen L. Cochi, Robin J. Biellik, Edward W. Brink, Olen M. Kew, Mark A. Pallansch, Walter A. Orenstein, and Alan R. Hinman. 1992. "Epidemiology of poliomyelitis in the United States one decade after the last reported case of indigenous wild virus-associated disease." *Clinical Infectious Diseases* 14(2):568–579. DOI: 10.1093/clinids/14.2.568.

Sydenstricker, Edgar. 1928. "The statistical evaluation of the results of social experiments in public health." *Journal of the American Statistical Association* 23(161):155–165. DOI: 10.2307/2277580.

Tanzi, M-L., P. Colotto, M. Vignali, P. Affanni, U. Bracchi, and E. Bellelli. 1997. "Antipoliomyelitis neutralizing antibodies in maternal and neonatal serum." *European Journal of Epidemiology* 13(5):559–565. DOI: 10.1023/A:1007345930234.

Trevelyan, Barry, Matthew Smallman-Raynor, and Andrew D. Cliff. 2005*a*. "The spatial dynamics of poliomyelitis in the United States: From epidemic emergence to vaccine-induced retreat, 1910–1971." *Annals of the Association of American Geographers* 95(2):269–293. DOI: 10.1111/j.1467-8306.2005.00460.x.

———. 2005*b*. "The spatial structure of epidemic emergence: Geographical aspects of poliomyelitis in north-eastern USA, July–October 1916." *Journal of the Royal Statistical Society, Series A* 168(4):701–722. DOI: 10.1111/j.1467-985X.2005.00372.x.

Troesken, Werner. 2001. "Race, disease, and the provision of water in American cities, 1889–1921." *Journal of Economic History* 61(3):750–776.

———. 2004. *Water, race, and disease*. MIT Press, Cambridge, MA.

Turner, Thomas B., David H. Hollander, Sonia Buckley, U. Pentti Kokko, and Charles P. Winsor. 1950. "Age incidence and seasonal development of neutralizing antibodies to Lansing poliomyelitis virus." *American Journal of Hygiene* 52(3):323. DOI: 10.1093/oxfordjournals.aje.a119427.

U.S. Department of Health, Education, and Welfare. 1956. "Death rates by age, race, and sex. United States, 1900–1953: Selected Causes." Vital Statistics – Special Reports 1–31, National Office of Vital Statistics, Washington DC.

U.S. Department of Health, Education, and Welfare. 1956. "Vital statistics of the United States, 1954: Volume II." Mortality data, National Office of Vital Statistics, Washington DC. http://www.cdc.gov/nchs/data/vsus/VSUS_1954_2.pdf.

———. 1957. "Vital statistics of the United States, 1955: Volume II." Mortality data, National Office of Vital Statistics, Washington DC. http://www.cdc.gov/nchs/data/vsus/VSUS_1955_2.pdf.

———. 1958. "Vital statistics of the United States, 1956: Volume II." Mortality data, National Office of Vital Statistics, Washington DC. http://www.cdc.gov/nchs/data/vsus/VSUS_1956_2.pdf.

———. 1959. "Vital statistics of the United States, 1957: Volume II." Mortality data, National Office of Vital Statistics, Washington DC. http://www.cdc.gov/nchs/data/vsus/VSUS_1957_2.pdf.

———. 1960. "Vital statistics of the United States, 1958: Volume II." Mortality data, National Office of Vital Statistics, Washington DC. http://www.cdc.gov/nchs/data/vsus/VSUS_1958_2.pdf.

van Panhuis, Willem G., John Grefenstette, Su Yon Jung, Nian Shong Chok, Anne Cross, Heather Eng, Bruce Y. Lee, Vladimir Zadorozhny, Shawn Brown, Derek Cummings, and Donald S. Burke. 2013. "Contagious diseases in the United States from 1888 to the present." *New England Journal of Medicine* 369(22):2152–2158. DOI: 10.1056/NEJMms1215400.

Van Riper, Hart E. 1947. "Poliomyelitis." *Journal of the American Medical Association* 135(2):74–76. DOI: 10.1001/jama.1947.02890020004002.

Whipple, George C. 1907. *The value of pure water*. John Wiley & Sons, New York.

———. 1908. *Thyphoid fever: Its causation, transmission, and prevention*. John Wiley & Sons, New York.

Zajacova, Anna and Sarah A. Burgard. 2013. "Healthier, wealthier, and wiser: A demonstration of compositional changes in aging cohorts due to selective mortality." *Population Research and Policy Review* 32(3):311–324. DOI: 10.1007/s11113-013-9273-x.

Zinkernagel, Rolf M. 2001. "Maternal antibodies, childhood infections, and autoimmune diseases." *New England Journal of Medicine* 345(18):1331–1335. DOI: 10.1056/NEJMra012493.

———. 2002. "Uncertainties — discrepancies in immunology." *Immunological Reviews* 185(1):103–125. DOI: 10.1034/j.1600-065X.2002.18511.x.